

EMERGENCIES IN
MEDICAL PRACTICE

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EMERGENCIES IN MEDICAL PRACTICE

FIFTH EDITION



E. & S. LIVINGSTONE, LTD.
EDINBURGH AND LONDON

1922

<i>First Edition</i>	-	-	-	1948
<i>Second Edition</i>	-	-	-	1950
<i>Third Edition</i>	-	-	-	1952
<i>Fourth Edition</i>	-	-	-	1954
<i>Fifth Edition</i>	-	-	-	1956
<i>German Translation</i>	-	-	-	1956
<i>Spanish Translation</i>	-	-	-	1956

Printed in Great Britain

PREFACE TO THE FIFTH EDITION

THE need for a new edition two years after its predecessor is a measure of the rapid progress of medicine and the demand for information of the kind this book aims to provide. All chapters have been thoroughly revised and those on Psychiatric Emergencies, Emergencies in Skin Disease and much of the chapter on Emergencies in Renal Disease have been completely rewritten. Details about drugs such as paraldehyde, aminophylline and vitamin K which are mentioned in many chapters have been collected into sections in the chapter on Practical Procedures. Throughout the book the opinions and personal practices of contributors have been stated rather than just the experience of others in order to avoid the danger of the book becoming, with each new edition, merely a review of the literature.

Helpful suggestions have been received from many doctors and these have been most valuable in bringing the work up to date. Further suggestions will be welcomed.

I would like to acknowledge the patient and skilful handling of the large amount of correspondence and typing by my secretary, Mrs. G. Butcher, and the preparation of many illustrations by Mr. F. Hardwicke Knight, a most accomplished photographer. Throughout the work Mr. Charles Macmillan's vast experience of medical publishing has been freely available to me. Finally, I would like to record my thanks to my wife and family for their forbearance during the long hours of editorial work in a small household and for being the willing subjects of some of the illustrations.

C. ALLAN BIRCH.

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CHAPTER I

The Emergency Bag

EVERY practitioner who is liable to be called in an emergency should have ready for immediate use an "emergency bag." Whether it is a combined medical and surgical bag will depend on the doctor and his type of practice. As experience and skill are at least as important as the contents of the bag, only the drugs and equipment with whose use he is familiar should be included (see also *Identification of Tablets*, page 511). A selection from the following list is suggested:—

Drugs.

Smelling salts.

Morphine 16 mg. (gr. $\frac{1}{4}$) in Ampins (Fig. 1) or 22 mg. (gr. $\frac{1}{3}$) in hypodermic tablets.

Nalorphine (Lethidrone) 10 mg. in 1 ml. ampoule (morphine antagonist see page 14).

Atropine hypodermic tablets 0.65 mg. (gr. $\frac{1}{100}$) (see page 358).

Injection of Nikethamide B.P. Three 2 ml. ampoules or Ampins.

Injection of Adrenaline B.P. in bottles, ampoules or Ampins.

Paraldehyde. Several 5 ml. ampoules.

Thiopentone sodium. Two 0.5 G. ampoules and eight 5 ml. ampoules of distilled water (*i.e.*, enough to make a 2.5 per cent. solution).

Fifty per cent. sterile solution of dextrose. 20 fl. oz.

Pethidine. Several ampoules containing 50 mg. in 1 ml.

Insulin. One 5 ml. bottle of soluble insulin (40 units per ml.).

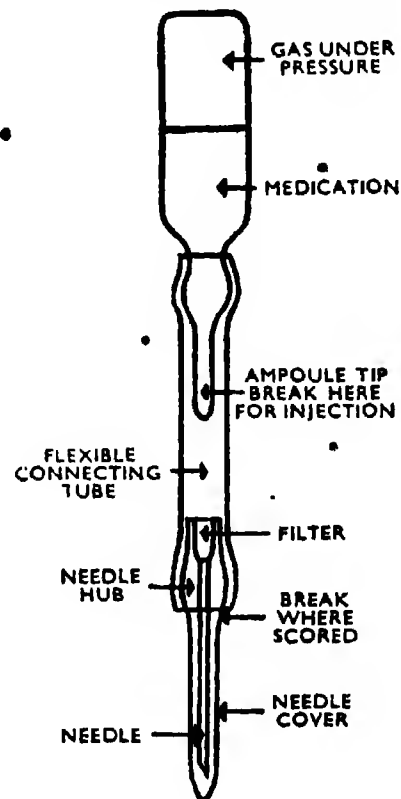


FIG. 1

THE AMPIN AUTOMATIC INJECTOR

Method of Use

Remove the needle cover. Insert the needle into the tissue.

Squeeze the plastic connecting tube and release. If the needle is in a vein blood will show on the filter pad.

If the aspiration test is negative break the ampoule tip by bending the plastic tube and the contents will be injected.

- /Tetanus antitoxin. Two ampoules of 3,000 units each.
- /Chloral hydrate solution. 4 fl. oz. of a solution containing 2 G. (30 gr.) to 15 ml. ($\frac{1}{2}$ fl. oz.) flavoured with syrup of ginger. Barbiturate tablets or capsules of the doctor's choice.
- Universal antidote (*page 5*).
- /Sulphonamide tablets (say sulphadimidine [Sulphamezathine]) —a small bottle.
- /Penicillin. Two vials of 500,000 units each and two vials of procaine benzyl penicillin.
- A supply of a "wide spectrum" antibiotic such as Tetracycline including paediatric drops.
- A bottle of antiseptic such as solution of chloroxylenol B.P. (Dettol) or surgical spirit.

DRUGS FOR OPHTHALMIC EMERGENCIES.

- Amethocaine drops 1 per cent. in drop bottle.
- Lamellæ of Homatropine B.P. 0.65 mg. (gr. $\frac{1}{100}$).
- Lamellæ of Fluorescein 0.26 mg. (gr. $\frac{1}{250}$).
- One tube containing 4 G. (60 grains) of each of the following eye ointments:—
- Eye Ointment of Atropine B.P. 1 per cent.
- Physostigmine Eye Ointment B.P. 1948. 1 per cent.

Appliances.

- A skin pencil.
- 1 ml., 2 ml. and 20 ml. Record syringes fitted with hypodermic and serum needles respectively and kept in 70 per cent. alcohol in spirit proof cases. (Alternatively in dry, sterile pack).
- Sterile water in clinbritic bottle.
- One teaspoon.
- Rubber tourniquet.
- Rubber catheter.
- Rubber gloves and three thin rubber finger stalls.
- Cotton wool, bandages, strapping and instruments for suturing.
- Two stomach tubes (adult and child) with funnel.
- Mouth gag and tongue forceps.
- Two lumbar puncture needles (Barker, Harris), dry, sterile, in tubes.

Sparklet carbon dioxide apparatus.

Venesection needle.

Anæsthetic mask.

A pharyngeal airway.

Two throat swabs.

Thermometer, stethoscope, sphygmomanometer and diagnostic set.

Two screw-top specimen bottles.

Two urgency orders and two copies of form A.1. (Mental Treatment Act, 1960) (*see page 227*). (Shaw & Sons, 7 Fetter Lane, E.C.4. Tel.: CENTral 8171).

The drugs and appliances required for emergencies in hospitals are chiefly those mentioned in Chapter XXVIII on Practical Procedures (*page 509*) and Appendix VII on drugs (*page 652*).

J. W. CHEETHAM.

CHAPTER II

Acute Poisoning

(For Acute Food Poisoning see page 71)

(For Poisoning on Board Ship see page 373)

WHILE the fact that a patient has been poisoned is generally obvious, this is not always so and hence it is important, when confronted with an obscure sudden illness, that the possibility of poisoning should cross one's mind before valuable time is lost. Poisons sometimes enter the body by unusual routes, *e.g.*, camphor poisoning has resulted from excessive rubbing with camphorated oil. Blood, albumin and sugar in the urine, and even extensor plantar responses do not necessarily mean that the illness is "natural" since poisons may be responsible.

If foul play is suspected and the patient is *in extremis* a dying declaration (page 492) should be taken.

It is more important to know what effects a poison is producing, and how it was taken, than to know exactly what the poison is. The old antidotal treatment of poisoning was often impracticable because the poison was sometimes unknown, or if known, its antidote was forgotten, or if remembered, not at hand. The two important principles in treatment are:—

- (1) To remove or neutralise the poison.
- (2) To keep the patient alive by dealing with the effects of poisoning.

REMOVAL OR NEUTRALISATION OF POISONS

Inhaled poisons.

Artificial respiration (page 543) is used as described under CO poisoning.

Swallowed poisons.

In children vomiting (from tickling the palate or apomorphine 3 to 7.5 mg. ($\frac{1}{20}$ to $\frac{1}{8}$ grain) subcutaneously) is often more effective in emptying the stomach than is the stomach tube. In adults, except in the case of poisoning by some corrosives (see page 7) the stomach should be washed out, taking precautions

against inhalation of stomach contents. Apomorphine need only be used in adults if a stomach tube is not available or if it is thought that stomach contents might block the tube. If gastric lavage can be done early, it should have priority over the use of an antidote. At the same time, there is something to be said for giving a dessert-spoonful of the following "Universal Antidote" while preparations are being made to wash out the stomach. As it is very light and fluffy it should be made into a paste before swallowing.

Powdered activated charcoal	2 parts
Magnesium oxide (or milk of magnesia)	...	1 part	
Tannic acid (or strong tea)	1 part

NOTE.—Ordinary charcoal, burnt toast, etc., is useless.

Most of the dose will be returned with the stomach washings and it is best not to leave any in the stomach lest the poison be absorbed from it in the intestine.

KEEPING THE PATIENT ALIVE

The life of the patient is immediately endangered by one or more of the following results of poisoning:—

- (1) Asphyxia.
- (2) Coma.
- (3) Dehydration and chloride loss.
- (4) Pain and shock.
- (5) Delirium and convulsions.

Each of these may demand prompt treatment irrespective of the poison responsible.

Asphyxia.

This may be chemical (as in CO poisoning), obstructive (as from inhaled vomit, inflammatory exudate and œdema), or due to depression of the respiratory centre as in coma. The essentials of treatment are:—

- (1) To maintain a good airway.
- (2) To apply artificial respiration (*see page 543*).
- (3) To administer oxygen.

It is often wise to call for the help of a skilled anaesthetist to combat asphyxia by such measures as artificial respiration by mechanical means, postural drainage and suction.

Coma. (*For other causes see page 187*)

(1) Maintain an airway, and perform artificial respiration if necessary. Coma is nearly always accompanied by some degree of asphyxia.

(2) An analeptic (amphetamine sulphate 25 mg. intramuscularly) may be given if respiration is failing but powerful ones like picrotoxin should be avoided.

(3) Unless the patient is taking fluid by mouth within eight hours, put up an intravenous saline drip.

(4) Give penicillin at the outset to prevent pneumonia.

Dehydration and chloride loss.

Profuse vomiting and diarrhoea will soon produce a state of collapse from dehydration and chloride loss. The urine output falls. The skin becomes lax and the eyes sunken. Thirst is intense and cramps occur.

It is best to give half-strength physiological saline (40 grains to one pint, or 0.45 G. in 100 ml.) sweetened with glucose, and flavoured with orange or lemon. A gallon (4½ litres approximately) may be needed. The drinking of fluid should go on in spite of vomiting. It is nearly always necessary, however, to give physiological saline intravenously.

Pain and shock.

These are symptoms of corrosive poisoning, and should be treated by giving morphine 15 mg. (gr. ¼) subcutaneously and fluids intravenously.

Delirium and convulsions.

These are not common symptoms of poisoning but when present they contribute to a fatal result. One of the easiest ways of quietening a violent patient is to inject intramuscularly into whatever part presents, 5 to 10 ml. of paraldehyde straight from the bottle (*see page 508*). Thiopentone as a 2.5 per cent. solution, i.e., 0.5 G. in 20 ml. of distilled water injected intravenously is quicker. Four or five ml. may be sufficient.

The above principles apply to all cases of acute poisoning. Corrosive poisoning and carbon monoxide, aspirin, barbiturate and methyl alcohol poisoning are mentioned in more detail because they are more commonly encountered. Poisoning by mercurial salts for which new and efficient treatment is available is described on page 265.

CORROSIVE POISONING

(1) Safeguard respiration. Call in the help of an anæsthetist. Tracheotomy (page 569), oxygen (page 569) and artificial respiration (page 543) may be needed.

(2) Try to find out what was taken. If Lysol or Carbolic Acid within the last 15 minutes, wash out the stomach. If acid give a thin paste of magnesia. Soapsuds, toothpaste or washing soda may be used instead. Bicarbonate is dangerous because of effervescence. If alkali was swallowed, give vinegar diluted 1 in 3. If in doubt as to what was swallowed give white of egg.

(3) Give morphine 15 mg. ($\frac{1}{4}$ grain) to an adult unless respiration is depressed.

(4) Preserve any vomit for examination.

CARBON MONOXIDE POISONING

Carbon monoxide (CO) is poisonous because, having 300 times the affinity of oxygen for haemoglobin, it produces chemical asphyxia by converting oxyhaemoglobin (HbO_2) to carboxyhaemoglobin (HbCO). CO also interferes with the release of oxygen from HbO_2 . The degree of gassing varies with the concentration, duration of exposure and reaction of the individual. Large persons are less affected by the same concentration than smaller persons, *i.e.*, children, since the rate of breathing depends on the surface area of the body but the blood volume varies as the weight of the body. The smaller the bulk of the body the greater the relative surface. That is why canaries are used as CO detectors. A man has only one twentieth of the skin area of a canary in relation to his body weight and so would need 20 times the period of exposure to be poisoned to the same degree. The reaction between CO and Hb is reversible. CO is eliminated at the rate of 30 to 50 per cent. per hour and more rapidly if pure oxygen is breathed. If a patient survives for 24 hours no CO will be left in his blood.

DIAGNOSIS.—CO poisoning is often obvious—as when a suicidal patient is found in a gas-filled room; but accidental poisoning may be very insidious. While 60 per cent. HbCO is necessary to cause coma in non-anaemic patients lower levels may be fatal in the decrepit, the diseased, the drugged and the drunk—the four D's of CO poisoning. It may occur from the CO evolved from a geyser in a small bathroom or from a bucket of hot coals or charcoal

burning in a restricted supply of oxygen. Persons affected by CO look very life-like. Respiration fails first but circulatory failure is not complete until some 12 minutes later. If gassing has been slight or if blood levels are low when coma persists then some other cause, *e.g.*, mixed poisoning or a cardio-vascular accident should be sought. For CO estimation heparinised blood covered with liquid paraffin should be sent. If there is any doubt, treat for CO poisoning by giving oxygen. This cannot harm the patient, whereas delay in its use might be serious in real CO poisoning.

RESCUE.—Is it safe to enter?

This question may not arise when the patient can be rescued quickly as from a gas oven but the rescuer should always go into the room crouching after taking a few deep breaths. (Coal gas, but not CO, is lighter than air). He should be "roped" so that he can be dragged out if overcome and does not come out after an agreed time, say one minute. The gas should be turned off and the patient dragged out by the heels.

Rescue from a difficult position, *e.g.*, a man-hole or bedroom should make one pause to consider the best method for it is foolish to rush in and become a casualty oneself particularly when it is unlikely that the patient can be rescued alive. The rescuer may lose the power in his legs while still conscious and so be unable to escape. A Pallado-Sulphite CO detector can be used, at a distance if necessary, to determine the concentration of CO. The atmosphere is drawn through a tube for two minutes and the length of colour change in it indicates the CO percentage. While a man can work indefinitely in a concentration of up to 10 in 100,000 (0.01 per cent.) a concentration of 200 parts per 100,000 (0.2 per cent.) is dangerous for exposures of up to one hour. A fatal concentration for exposure of less than one hour is 400 per 100,000 (0.4 per cent.). It must not be forgotten that the atmosphere may be low in oxygen and that other poisonous gases may be present also. For difficult and prolonged rescue attempts a fresh air respirator or a self-contained oxygen apparatus should be worn (*page 542*).

TREATMENT AFTER RESCUE.—Clear the airway by pulling forward the tongue and using a swab. Beware of injuring the tongue with the crushing type of tongue forceps as it may swell rapidly. If breathing is very shallow, apply artificial respiration preferably in the prone position (Holger Nielsen method, *page 544*). Because

of the risk of CO_2 retention when the respiratory centre is depressed it is at present recommended by the Medical Research Council that CO_2 should not be administered with O_2 in the resuscitation of patients receiving artificial respiration. Some feel however, that CO poisoning is a special case and that stimulation of respiration by CO_2 may be advantageous in increasing the rate of CO excretion and preventing late sequelae. Pending further experimental evidence no definite lead can be given and whether to use CO_2 in an individual case remains a question for the physician concerned to decide. Oxygen should be given through an oro-nasal mask or if necessary by a breathing machine (*page 559*). The position of the limbs should be carefully watched, for the combination of anoxia and vascular obstruction may quickly lead to gangrene.

ASPIRIN POISONING

Aspirin has become a domestic panacea and poisoning by it is increasingly common. The dehydration, low blood pressure, over-breathing, positive ferric chloride test and partial reduction of Fehling's solution produce a picture resembling diabetic coma. Tinnitus, deafness and skin rashes and the history point to aspirin as the cause. Dehydration may be so intense as to soak the mattress completely and must be made up by giving intravenous fluid, bearing in mind the danger of a too copious infusion (*see Circulatory Overloading, page 48*).

The salicylate radical which aspirin yields on hydrolysis causes increased depth of respiration by a central action ("salicylate dyspnoea") and by "washing out" CO_2 causes a respiratory alkalosis. It would seem wrong, therefore, to give alkali in aspirin poisoning and the large doses of it formerly recommended (roughly equivalent in weight to the aspirin swallowed) have been found to cause tetany. It is true, however, that alkalis sufficient to make the urine alkaline hasten the removal of salicylates from the blood. The proper course would therefore be to give sodium bicarbonate in amounts just sufficient to keep the urine alkaline. If it has to be given intravenously, say 50 G. per litre, it should be remembered that it cannot be sterilised by boiling, as the bicarbonate decomposes. If bicarbonate is added to sterile water under sterile conditions, the solution is safe for intravenous use. Samples of urine should be obtained by means of a self-retaining catheter and tested frequently for reaction and intensity of the ferric chloride reaction.

Warmth, oxygen and other measures, as described under "Medical Shock" (page 144), may be used as indicated.

Two further urgent symptoms might arise in aspirin poisoning. A violent paroxysm of asthma might be caused in an aspirin-sensitive asthmatic. Hæmatemesis is also a possibility because aspirin is a gastric irritant and also decreases the coagulability of the blood.

BARBITURATE POISONING

This usually presents as coma with flaccid limbs, and the plantar reflexes may be extensor. An empty labelled bottle or bottles may point to the diagnosis and be helpful in those cases in which more than one poison has been swallowed. A box of the various sedative tablets shown to the relatives may help them to identify the one taken. The effects of two or more sedatives are at least additive and one may even potentiate the other. In doubtful cases urine, blood and vomited or aspirated gastric content should be tested for barbiturates (a laboratory procedure). Senility, associated disease, shock and anoxia from pulmonary œdema and suffocation all play additional parts in determining the outcome.

Treatment.—Evaluation of the depth of coma is the most important factor determining treatment but there is no rule of thumb method and reliance cannot be put on any one sign (e.g., absent corneal reflexes) alone. Overdoses of short-acting barbiturates (Amytal, Nembutal and Seconal) produce deep coma quickly whereas slower acting ones (phenobarbitone) cause less deep coma of slower onset. Many cases are mild and the overall mortality is under 10 per cent. By summing up the evidence the doctor should decide whether poisoning is mild (patient conscious), moderate (patient stuporose), or severe (patient comatose with low blood pressure, subnormal temperature and absent tendon reflexes).

The principles of treatment are to maintain an airway; to remove any poison still in the stomach; to use a barbiturate antagonist and to prevent pneumonia. The order in which the following measures are used will depend on the circumstances of the case.

(1) **Transfer the patient to hospital at once.** Travel with him or send someone capable of performing artificial respiration. Pending removal and if still conscious an emetic (2 tablespoonsful of

salt in 5 fl. oz. of water) may be given by mouth. It acts more quickly when sitting up than when lying down. Apomorphine 6 mg. ($\frac{1}{10}$ grain) may be used instead but is inadvisable if the patient is deeply comatose.

(2) **Gastric lavage.** The stomach should first be emptied by a tube and syringe or a Senoran's evacuator (Fig. 541). In deeply comatose patients gastric lavage can be hazardous (page 35) and it is wise to call in the anæsthetist so that a cuffed endo-tracheal tube can be passed first. Water (2 gallons in all) is best for lavage and bicarbonate solution should be avoided as it increases the solubility of barbiturates. If coffee (1 pint) or White Mixture of Magnesium Sulphate B.P.C. (2 fl. oz.) is left in the stomach the patient must be watched carefully afterwards lest it be regurgitated and inhaled (page 35).

(3) **Maintain an adequate airway and use artificial respiration** (page 543) and oxygen (page 569) as the state of the patient indicates. Careful bronchoscopic suction may be needed.

(4) **Bemegride, (Megimide. Methyl Ethyl Glutarimide).** This barbiturate antagonist avoids the risk of prolonged deep coma and will bring the patient to a "safe state" of light anæsthesia from which recovery will readily occur. It is best injected into the tubing (for risk of air embolism see page 26) of an intravenous drip giving 50 mg. (= 10 ml.) every five minutes. Follow each injection in the same way by 15 mg. of Amiphenazole (Daptazole), a respiratory stimulant, freshly dissolved. Groaning, movement, and return of tone and reflexes indicate attainment of the "safe state." This occurs after about two hours treatment. The total dose of Bemegride (Megimide) is usually about 1 G. and of Amiphenazole (Daptazole) 0.3 G. Relapse may call for further dosage. The drugs should not be used to attain full awakening. Vomiting and retching are early signs of over-dosage.

(5) **Fluids.** If because of large dosage, mixed poisoning or some other cause, the patient is comatose for a long time, fluid by intravenous or intragastric drip will be needed. A self-retaining catheter should also be passed.

(6) **Colonic lavage.** This may remove unabsorbed tablets and should always be used together with a purgative (see paragraph 2) if delayed acting tablets have been swallowed.

(7) **Antibiotics.** Give penicillin 500,000 units 8 hourly and turn the patient frequently.

(8) **Vitamin B₁₂** 1,500 µg. and **A.C.T.H.** 20 units in 1,000 ml. of physiological saline have been used with success and have experimental support. As they are without danger they might be tried if response to other treatment is poor. In any given case clinical findings such as pulmonary oedema and very low blood pressure, and laboratory findings such as the blood urea, plasma bicarbonate and serum and urinary chlorides may call for special measures. For example, 1-noradrenaline (Levophed) 2 mg. may have to be added to 500 ml. of intravenous infusion to correct hypotension.

(9) In case of hypothermia a **heat cage** should be used to restore body temperature.

OUTMODED TREATMENT.

1. **Strong Analeptics (Picrotoxin)** are out of favour because they may cause convulsions and in any case only achieve by respiratory stimulation what can more safely be achieved by artificial respiration.

2. **Lumbar puncture.** This is of little value as only low concentrations of barbiturates are found in the C.S.F.

3. **Forced Diuresis.** High rates of urine flow do not significantly increase the renal clearance of barbiturates. The idea of "blood lavage" by parenteral fluid and mercurial diuretics should be regarded with reserve.

METHYL ALCOHOL (METHANOL) POISONING

This usually results from drinking methylated spirit but it may follow its use on the skin. Methanol itself is toxic and is slowly oxidised to formic acid which is more toxic. Individual tolerance varies greatly and depends partly on whether food and ethyl alcohol were also taken.

Symptoms result partly from the direct effect of methanol and its products and partly from acidosis. They are usually delayed for up to 24 hours. The poison is distributed largely in proportion to the water content of tissues. Hence the intra-ocular fluids are greatly involved. Mild cases resemble ordinary alcoholism, but in more serious ones there is epigastric pain, vomiting, blindness and delirium going on to prolonged coma. Some confusion may result from the fact that formic acid in the urine will reduce Fehling's solution.

Treatment.—After gastric lavage (*see page 537*) pectin should be given since it prevents the oxidation of methyl alcohol. Measures to combat acidosis are most important. If laboratory help is available the amount of alkali needed can be determined from the CO_2 combining power of the plasma (" CO_2 capacity "—normal range for venous blood 55 to 77 volumes per 100 ml.) by the formula $\text{Kg. body weight} \times 0.026 \times (65 - \text{CO}_2 \text{ combining power}) = \text{grammes of sodium bicarbonate to bring } \text{CO}_2 \text{ combining power back to 65}$. The arterial blood pH is a finer guide. (For arterial puncture *see page 579*). In the absence of laboratory help 10 G. of sodium bicarbonate should be given every 2 hours until the urine is alkaline to litmus and then enough to maintain it so. In severe cases cerebro-spinal fluid should be removed. It is wise to cover the eyes until the danger of blindness is past. Ethyl alcohol inhibits the formation of formic acid by competing with the enzyme system involved in the oxidation of methyl alcohol. It is therefore recommended to give small quantities of whisky every four hours.

SPECIAL TREATMENTS FOR CERTAIN POISONS

If the poison is known and there is a good antidote, this may be used *when the general measures to eliminate the poison and to keep the patient alive have been started*.

"Home Perm" Solution.

Outfits for hair waving at home contain a powder (potassium bromate) from which to make a neutralising solution to apply, after the first one. It looks like water and is almost tasteless and so may be drunk in error. Should this happen, sodium thiosulphate (photographic "hypo") 1 teaspoonful should be given by mouth and 10 to 50 ml. of a 10 per cent. solution injected intravenously.

Aniline dyes.

These cause grey cyanosis from methæmoglobinæmia. Give methylene blue 2 mg. per Kg. body weight as a 1 per cent. solution by slow intravenous injection.

Arsenic (weed killer).

While preparing to wash out the stomach give a mixture containing 30 ml. of solution of ferric chloride and 30 G. of sodium bicarbonate in 120 ml. of water. (Add the bicarbonate bit by bit and allow each effervescence to subside. The mud-like resulting

mixture contains ferric hydrate which with the arsenical stomach contents forms almost insoluble ferric arsenite). BAL (Dimercaprol) (page 265) should also be used.

Paraffin oil (kerosene) and petrol.

When vapour is absorbed from the lungs or liquid from the stomach the effects on the nervous system resemble those of alcohol and recovery can occur. Even small amounts of liquid kerosene, however, can cause serious pneumonia. Special care should therefore be taken during gastric lavage lest some kerosene enters the lungs.

Mercury (Corrosive Sublimate).

Give BAL (Dimercaprol) (page 265).

Cyanide (see page 346).

Give amyl nitrite to inhale for 30 seconds every 2 minutes and also sodium nitrite 0.5 G. in 10 ml. of water by mouth.

Ferrous sulphate.

Pleasantly coated tablets of ferrous sulphate may be swallowed in large numbers by a small child. They cause corrosion of the gastric mucosa followed by absorption of toxic amounts of iron. Gastric lavage followed by a bismuth mixture should be used. Dimercaprol (BAL) is contraindicated because the BAL-iron complex is more toxic than iron itself. α -Tocopherol (Ephynal, Roche), 100 mg. daily by mouth or intramuscularly, is recommended.

Ethylene glycol ("Anti-freeze").

This is oxidised to oxalic acid and so calcium gluconate 10 ml. of a 10 per cent. solution should be given slowly by intravenous injection to convert oxalic acid to the relatively inert calcium oxalate.

Morphine. Pethidine. Physeptone.

If swallowed, wash out the stomach with 1 per cent. potassium permanganate (a saturated solution is 5 per cent.). Nalorphine (Lethidrone) is the specific antidote but an imperfect one for it is a near relative of morphine and while countering respiratory depression it may increase sedation and vomiting. It combines with the receptors on which morphine, etc., act and prevents or reverses their action. Ampoules contain 10 mg. in 1 ml. and the usual dose by intravenous injection is 10 to 40 mg. *It is useless against barbiturates.*

Naphthalene and para-dichlorobenzene. (Moth balls).

These are not very toxic. Treatment should be on general lines by gastric lavage. Castor oil in which they are soluble should be avoided. Serious cases with hæmolytic should be given a blood transfusion and cortisone.

DDT (Dicophane B.P.) poisoning.

The minute quantities consumed in food are innocuous but if a large amount of DDT is ingested it affects the nervous system and causes excitation, tremors, convulsions and paralysis. Treatment is by gastric lavage and purgation. The poison is soluble in fats and so these should be avoided until it is all excreted in about two weeks or until the urine is free from poison.

Insecticide poisoning (*see page 356*).

C. ALLAN BIRCH

CHAPTER III

The Hazards of Medical Procedures

Even the most simple procedures can sometimes be hazardous. It is a good habit therefore to remind oneself before starting of what might go wrong and to know what one would do if it did.

ACCIDENTS WITH NEEDLES

Broken needles.

SHORT needles usually break at the junction of the shaft and the butt. Long needles may rust internally and break anywhere, especially if they have been in contact with iodine. Nickel needles bend rather than break and may be used for lumbar puncture if sudden jerks are expected. All needles should be tested before use and a needle should never be inserted as far as it will go. A guard may be placed on a needle to prevent too deep insertion as in liver biopsy. Should a needle break in the tissues and sufficient project, it may be removed by forceps, but more often no part remains visible though pressure on each side of the puncture may reveal the end. Beware of making a small incision and poking about. Prevent the patient from moving the part; tell him what has happened but do not admit negligence or offer compensation. Have the needle removed at a planned operation. The electromagnet may help but incision will still be necessary. If the patient is too ill, say nothing and leave him alone. He may die. If he recovers from his illness, always have him X-rayed before exploring. Small portions of the needle have been left in the body and even in the pleural cavity without causing untoward effects, and pieces of dental needles in the pterygoid fossa are usually best left there. In all cases the doctor would be wise to report the circumstances and send the pieces of needle to his medical protection society.

Venepuncture.

This not uncommonly causes fainting which may be avoided by a few points of detail. Let the patient be seated and his arm comfortably supported on a pillow. Don't use the word "blood" or let the patient see the needle. Don't pinch the skin with the tourniquet. Don't let antiseptic trickle on the arm—he will think it is blood. Use a sharp needle but don't probe for the vein. It

is there if you will feel for it. If, in spite of all this, the patient faints, lower his head quickly.

Extra-venous injections.

Many substances commonly given intravenously, such as thiopentone, calcium chloride and the soluble sulphonamides (except Soluthiazole, May and Baker, *see page 600*) are highly irritant if they leak into the subcutaneous tissues. Diodone (*see page 654*) and the newer substances used for excretion urograms (Hypaque, Bayer and Urografin, Schering) have the advantage of being non-irritant subcutaneously. Sodium acetrizate (Diaginol, May and Baker; Urokon, Bayer) is slightly irritant and is best avoided by the intravenous route. In children up to five years 5 ml. of Hypaque into each buttock with or without hyaluronidase 1,500 international units (*page 654*) will give good pyclograms.

When intravenous injection is likely to be difficult because of poor veins, the following measures should be adopted to ensure that the veins will be dilated. Flicking the vein with the finger will often make it dilate. Failing this the whole arm should be wrapped in a warm moist towel and hot water bottles applied for twenty to thirty minutes. Venous spasm is more likely in peripheral (especially leg) veins than in central ones because the muscle in their walls increases the further they are from the heart. As cold and pain may cause venous spasm, ether on the skin should be avoided and the subcutaneous tissues down to the vein should be infiltrated with 1 per. cent. procaine. A glass adaptor between the syringe and the needle (Fig. 2) makes it easy to see the blood on withdrawing the piston and thus indicates that the vein has been entered. This is otherwise impossible when using dark-coloured solutions.

If the solution is dark in colour so that the extent of the leakage can be seen, it is good practice to excise the area, irrigate

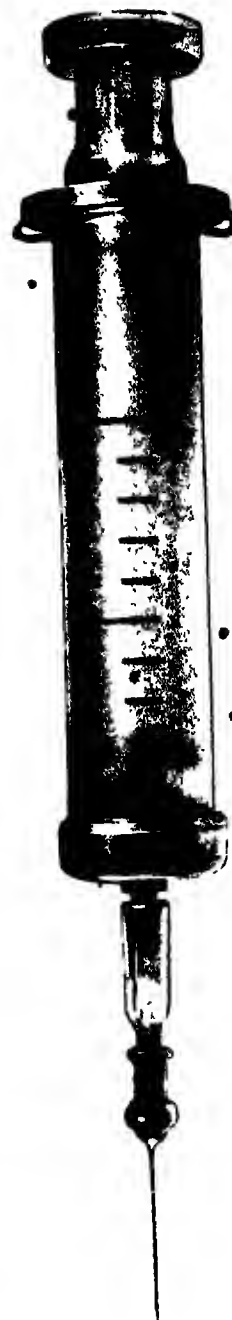


FIG. 2
Glass adaptor
between syringe
and needle.

with physiological saline and stitch it up. In the case of small leaks of pale solutions an attempt to aspirate may be made. Injection of a little 1 per cent. procaine with hyaluronidase (*page 654*) may prevent tissue damage from thiopentone. The limb should be immobilised and warmth applied.

Entry of an artery instead of a vein.

This rare accident may happen when a tight tourniquet has obliterated pulsation in an aberrant artery lying between the fascia and the skin so that it looks and feels like a vein. It may also happen when, in exploring a plump ante-cubital fossa, a vein is transfixed and a deeper artery entered. It would be safer to use a vein on the dorsum of the hand. In resuscitating the newborn it is thought that nikethamide solution intended for the umbilical vein (which is collapsed in asphyxia) might be injected into one of the umbilical arteries. It could then reach the sciatic branch of the internal iliac artery—the main supply to the leg in the foetus—and would account for cases of sciatic paralysis and gangrene of the buttock in the newborn. Two simple precautions should indicate that the limb vessel in question is an artery and not a vein. Palpation *before applying a tourniquet* will reveal pulsation and the blood withdrawn will generally be bright red. Although thiopentone may cause venous blood to darken a little in the syringe it will not cause arterial blood to look like venous blood. Another point which should suggest in the case of thiopentone that the injection is intra-arterial is that the onset of narcosis will be delayed. It is always wise to wait a few seconds after commencing a supposed intra-venous injection and to proceed only if no untoward effects appear.

The intima of arteries is specially sensitive to alkaline solutions such as thiopentone which if injected intra-arterially generally cause intense burning pain. This may be absent, however, and then the only immediate signs are coldness and blanching of the hand followed by urticaria. Subsequent spasm, thrombosis and oedema may lead to gangrene. Hexobarbitone soluble is non-irritant and almost equally as effective as thiopentone.

Treatment.—Since the accident may have such serious consequences prompt treatment is indicated. The objects are to cause full vasodilatation, to prevent thrombosis and to relieve pain.

1. If you have sufficient presence of mind don't remove the needle but inject into the artery at once (before blood clots in the

needle) 5 ml. of 2 per cent. procaine. This is likely to be at hand but tolazoline (Priscol. Ciba) 10 to 20 mg. in 2.5 per cent. solution is better. Follow this with the first dose of heparin (page 595) preferably into the artery so that it may act on blood trapped there.

2. Give Morphine 16 mg. (gr. $\frac{1}{4}$) or Pethidine 100 mg. into the other arm.

3. Ask the anaesthetist (he is probably there already) to perform brachial plexus block. Since the patient (because of thio-pentone) will be unable to co-operate at least 20 ml. of anaesthetic solution with 1,500 international units of hyaluronidase (page 654) should be used. When the block is completed the subclavian artery should be punctured and 50 mg. of tolazoline (Priscol. Ciba) with 80 mg. of papaverine in physiological saline injected into it. These procedures may be repeated several times if necessary.

4. Arrange for anticoagulant therapy to continue for several days.

5. Ensure warmth as advised for peripheral embolism (page 167).

If operation is not urgent it should be postponed but heparin should not be omitted on the grounds that operation is imperative.

Chest pain following intravenous injections.

Anginal pain may be precipitated in patients with coronary insufficiency when too rapid injection particularly of iron and calcium preparations causes a fall of blood pressure. Even normal subjects may experience substernal discomfort. All therapeutic intravenous injections should be given slowly.

Puncture of the trachea.

The trachea may be damaged in an attempt to take blood from the internal jugular vein of an infant. Subcutaneous and mediastinal emphysema and pneumothorax may result and cause death.

Bone marrow infusion.

This shares many of the risks of other forms of parenteral infusion. A special risk of using the sternal marrow is that of entering the anterior mediastinum or even the pleural spaces. It has happened that the first insertion of the needle has been deemed unsatisfactory when it had, in fact, perforated the posterior plate, so that infusion at another nearby site caused much fluid

to pass through the previously-made hole. These difficulties may be avoided by using a trephine needle. In infants, the tibial route is safer and more practicable than the sternal.

Pericardial hæmorrhage.

This is one of the remote risks of intracardiac injection of drugs, such as adrenaline (for cardiac arrest). It may also occur, but is less likely when tapping a pericardial effusion by the anterior route. If blood is obtained when doing this there may be doubt

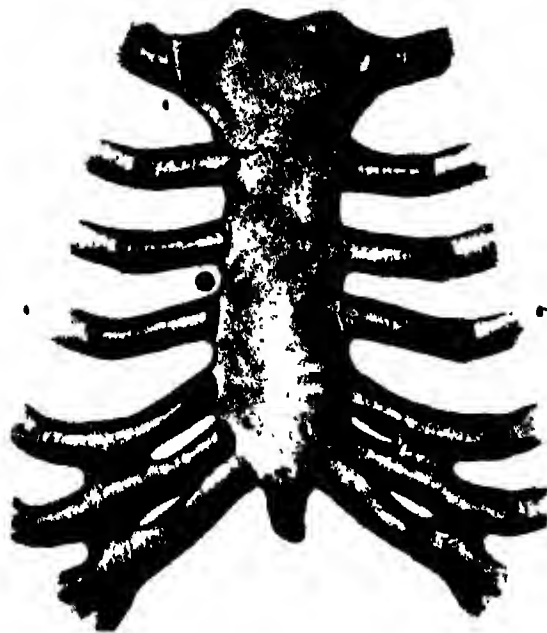


FIG. 3
Showing entry point for injection of
right auricle.
(Hamilton Bailey—*Emergency Surgery*.)

as to whether a ventricle has been entered. It is possible to make sure by leaving the needle in position and injecting saccharine (*see page 127*). If the patient tastes this (almost immediately from the left ventricle or in seventeen or more seconds from the right ventricle) it means that the needle has entered a ventricle. Pressure of blood in the pericardial sac rather than its total amount is responsible for "cardiac tamponade" and as little as 200 ml. has caused death.

Hæmopericardium from puncture is less likely to occur when the auricles, rather than the ventricles, are injured. As the auricles are also more sensitive to mechanical stimuli than the ventricles, needling and injection are more effective and less dangerous if an auricle is entered. The right auricle should be

chosen. A special curved needle 12.5 cm. (5 inches) long (Fig. 59, page 448) or failing this a lumbar puncture needle should be inserted in the third interspace at the upper border of the fourth rib close to the sternum and directed downwards and towards the middle line (Fig. 3). The auricle is within 5 cm. (2 inches) of the outer surface of the sternum in children but may be 11.25 cm. ($4\frac{1}{2}$ inches) deep in adults.

Lumbar puncture. • (*For technique see page 522*)

Too much preliminary prodding of the back may precipitate hysterical phenomena in susceptible patients as may also the painful periosteal injury of clumsy manipulation.

When meningitis is thought to have resulted from lumbar puncture the organism should be carefully sought and its sensitivities tested as it may be a resistant one which has escaped sterilisation.

Permanent paralysis has followed lumbar puncture in a few cases. It may have been caused by intrathecal bleeding, though this is usually harmless or causes only transient root irritation. A more probable cause is progression of a pre-existing lesion. Hence it is important to record carefully the physical signs present before puncture.

Insertion of the needle too far may damage or infect an inter-vertebral disc. This may be avoided by tilting the needle towards the patient's head. In this position it will encounter the next vertebral body above and not the disc. Lumbar puncture higher than the space between the third and fourth lumbar vertebrae should be avoided as a spinal cord extending lower than normal is not unusual.

Removal of C.S.F. may, by vascular dilatation initiate intracerebral or meningeal haemorrhage from diseased vessels. External rectus palsy may result from stretching of the sixth nerve from sudden alteration in intracranial dynamics.

Lumbar puncture is said to be dangerous in a patient with raised intracranial pressure from a subtentorial tumour because herniation of the medulla into the foramen magnum ("foraminal crowding") may result. Similarly in cases of supratentorial tumour, herniation of the uncus of the hippocampal gyrus into the tentorial hiatus may occur. The accident is something of a clinical ghost, however, and in only one of 401 cases of cerebral tumour

did lumbar puncture cause any untoward effects and even then the relationship was doubtful. Withdrawal of minimal volumes of C.S.F. provides no guarantee that this complication will not supervene, as leakage through the meningeal puncture may continue for some hours. Warning signals are an abrupt cessation of fluid flow, followed by neck pain and dysphagia. Respiratory failure and death follow in minutes or hours. Similar sudden collapse without lumbar puncture may occur in patients with raised intracranial pressure and emphasizes the precarious position of these patients.

Prevention of this accident lies in removing as little fluid as possible. A Dattner (double) needle is specially useful in doubtful cases (*page 526*). It is advisable to pierce the ligamentum flavum with the bevel of the needle in the spinal axis so that fibres are separated rather than cut across. The puncture will then close more easily. The "pencil point" needle with a hole at the side has the same object.

If collapse should follow lumbar puncture before the needle is withdrawn physiological saline equal in volume to the fluid removed should be injected. If the needle has been withdrawn raise the foot of the bed and give 2 ml. of nikethamide by slow intravenous injection. Start artificial respiration if need be. Call a neurosurgeon as ventricular tapping may be needed. While awaiting this give 500 ml. of 50 per cent. sucrose intravenously.

Cisternal puncture. (*For technique see page 526*)

An emergency may follow cisternal puncture if the needle is inserted more than 2 cm. after the occipito-atlantal ligament is pierced. In this case the medulla is injured—usually with fatal results. Deviation of more than 18° from the mid line may result in damage to a vertebral artery, with resulting subarachnoid hæmorrhage. This may also be due to damage to abnormal vessels inside the foramen magnum. As these are not uncommon, cisternal puncture is best avoided unless clearly indicated. In either of these emergencies little can be done, beyond the giving of analeptics (*see page 608*).

Marrow biopsy.

Marrow biopsy by sternal puncture presents few risks, but some accidents have occurred. A very ill patient might die from a cardiac inhibitory reflex caused by the puncture and to avoid this, it is wise to give a sedative first. Perforation of the sternum

particularly if the inner plate is softened by some neoplastic disease has led to death from hæmopericardium (*see page 20*). Hæmorrhage might also occur if the patient is suffering from a hæmorrhagic disease. Blood transfusion and vitamin K therapy (*page 599*) would be wise in such cases. The use of the iliac crest or a lumbar vertebral spine seems preferable to the sternum.

Liver biopsy.

Needle biopsy of the liver may be complicated by hæmorrhage and necessitate blood transfusion. It should be avoided if prothrombin activity is 50 per cent. of normal or less or if the platelet count is less than 100,000 per cu. mm. A preliminary course of vitamin K (*page 599*) is advised. Unless the patient controls his breathing by practice, the needle, fixed between the ribs, may tear the capsule as the liver moves. This is less likely when the puncture is made from behind. Puncture through the abdominal wall may avoid this risk but should not be done unless the liver is palpable. It is best to limit the depth of liver puncture to 5 cm. since beyond this a large branch of the portal vein may be entered. Incidentally, aspiration of much blood makes search for the bit of liver difficult. Needle biopsy should be avoided in obstructive jaundice because of the risk of puncturing a dilated biliary duct with resulting bile peritonitis or even bile embolism of the lungs. It is, of course, contra-indicated when there is infection in the pathway of the needle. The mortality rate in 20,000 punctures was 0·17 per cent.

EMERGENCIES FOLLOWING LOCAL ANALGESIA

(*For Emergencies in General Anaesthesia see Chapter XXIV*)

A fainting or syncopal attack from emotional causes may quickly follow the injection of procaine or, should it be inadvertently intravenous, from the adrenaline it contains. The aspiration test should be repeatedly used during subcutaneous infiltration lest a small vein be entered.

Symptoms of cerebral origin are less quick in onset. Occasionally unconsciousness is the first sign that anything is wrong. Slighter symptoms such as talkativeness, sweating and restlessness are common and may be overlooked or put down to "hysteria". They are due to cortical stimulation. Facial twitchings and dilated pupils follow and in severe cases go on to delirium, convulsions and respiratory failure. A local type of reaction may occur in the

larynx when local analgesia is followed by intense spasm of the cords.

PREVENTION.—Cocaine should never be injected but faulty labelling has caused it to be used in error. It has been largely replaced by procaine (for injection) and amethocaine or butacaine sulphate B.P.C. (Butyn, Abbott) (for surface application).

Toxicity of local analgesics increases in geometrical, not arithmetical progression, with increase in concentration. Hence a 2 per cent. solution is sixteen times more toxic than a 0.5 per cent. solution. The weakest practicable solution should, therefore, be used. The limits of dosage are variable but the total dose of cocaine hydrochloride should not exceed 60 to 100 mg. (1 to $1\frac{1}{2}$ grains). The maximum safe dose of procaine hydrochloride by injection is 1 gramme but it is wise to use less. 50 ml. of a 1 per cent. solution or 100 ml. of a 0.5 per cent. solution are usually sufficient. Amethocaine is four times more toxic than cocaine but its effective dose is only one-tenth that of cocaine. The total dose of amethocaine should not exceed 80 mg. (4 ml. of a 2 per cent. solution). Butacaine sulphate B.P.C. (Butyn, Abbott) 2 per cent. is probably the safest surface analgesic. All these figures refer to adults; children are less tolerant. Aged, shocked, myxoedematous and vitamin C-starved patients need less. Concentration of solutions by evaporation or cracked ampoules (*page 38*) should be borne in mind.

Too rapid absorption can be avoided by adding adrenaline to local analgesic solutions, *e.g.*, Injection of Procaine and Adrenaline B.P. which contains 2 per cent. w/v of procaine hydrochloride (2 ml. of solution of adrenaline hydrochloride in 100 ml. of procaine solution). This gives a concentration of adrenaline of 1 in 50,000 but concentrations of only 1 in 250,000 are used when large amounts of solution are injected. Absorption is rapid from surfaces covered by other than stratified epithelium. Cocaine should always be avoided for surface analgesia except in the nose, throat and eyes. Urethral injection of it is specially dangerous and more so if a stricture is present or trauma has occurred. For pharyngeal analgesia by gargling it is better to tell the patient to "gargle and spit" rather than to "gargle and swallow".

Patients with myasthenia gravis are specially liable to collapse after injection of procaine or cocaine.

A preliminary dose of a barbiturate is a wise precaution in nervous patients. When allergy is suspected a little of the drug may be applied intranasally to see if any reaction occurs.

Treatment.—For a syncopal attack lowering the head, smelling salts and an injection of nikethamide will suffice. If there is idiosyncrasy to procaine 0.5 mg. neostigmine (Prostigmin) intramuscularly or intravenously will quickly improve the patient. Convulsions should be cut short by the slow intravenous injection of 2.5 per cent. thiopentone (Pentothal): 5 to 10 ml. are usually enough. Collapse and coma would call for the use of artificial respiration (see page 543) oxygen administered by a mask (see page 573) and injection of picrotoxin or leptazol (see page 609). When death from respiratory depression seems imminent, 100 mg. ($1\frac{1}{2}$ grains) of ephedrine hydrochloride should be injected intravenously. If this fails the injection into the cisterna magna (see page 526) of 60 mg. (1 grain) of ephedrine hydrochloride in a few ml. of physiological saline has been recommended.

Always tell the patient what happened so that he can warn the doctor the next time.

EMERGENCIES ARISING DURING PNEUMOTHORAX AND PNEUMOPERITONEUM THERAPY

Pleural shock.

In rare cases collapse and even death have followed immediately on puncture of the pleura. This has been attributed to pleural shock but in many cases so diagnosed, the symptoms have clearly been those of cerebral or coronary air embolism.

Treatment.—Nothing useful can be done in such a case, but in the less severe cases which resemble vaso-vagal faints, smelling salts, brandy, and leptazol (see page 609), may be used. The head should be placed low. Mild cases showing transient faintness are not uncommon.

Hæmoptysis.

This means that the lung has been punctured as shown by an equal manometric swing on each side of zero. Usually, nothing more than reassurance is required.

Mediastinal and subcutaneous emphysema.

During artificial pneumothorax therapy, the needle may damage a pulmonary alveolus and cause interstitial emphysema

of the lung. The air may reach the mediastinum and rupture into the pleura causing a tension pneumothorax (*page 137*). The reverse of this phenomenon (pneumothorax rupturing into the mediastinum) does not occur. Mediastinal emphysema (*page 140*) is occasionally a complication of bronchoscopy.

Subcutaneous emphysema, localised to the site of the puncture does not cause urgent symptoms. When present in the neck it means that mediastinal emphysema is also present (*page 140*). Urgent symptoms are rare because escape of air into the neck relieves the mediastinal pressure. Air may spread to the retro-peritoneal tissues and cause abdominal pain. No treatment beyond the use of analgesics is required.

Pneumoperitoneum.

Rarely, this may be induced inadvertently if the pneumothorax needle is inserted too low. Failure to obtain a satisfactory manometric swing, and the disappearance of liver dullness should indicate what has happened. There may be pain in one or both shoulders, but if the phrenic nerve has been previously crushed, pain will be confined to the opposite side. No special measures are indicated.

When inducing therapeutic pneumoperitoneum, the bowel has, on a rare occasion, been injured, as shown by the presence of faecal matter in the needle. No harm results, and no treatment is indicated, but a period of observation is advisable. Pulmonary air embolism has occurred from liver injury. This can be more readily avoided by inserting the needle under the left costal margin. Coughing several days after a refill has been followed by air embolism, presumably from rupture of a venule.

Pneumothorax, generally right-sided, has occasionally complicated pneumoperitoneum therapy either immediately from congenital diaphragmatic defects or later from diaphragmatic rupture. Another route is via the mediastinum (mediastinal emphysema, *page 140*) with secondary rupture into both pleural cavities.

AIR EMBOLISM

Air embolism is of two clinical types—venous (or pulmonary) and arterial.

Venous (or pulmonary) air embolism.

This accident is most likely to be fatal if a large quantity of air enters a large vein near the heart, as when a poorly filled vein

is incised and held open by retraction of the edges of the wound. This mechanism is responsible for the very rapid death which sometimes follows a suicidal throat wound. During neck and axillary operations, it is less likely to occur if the head is low, so that the veins are distended. It should be

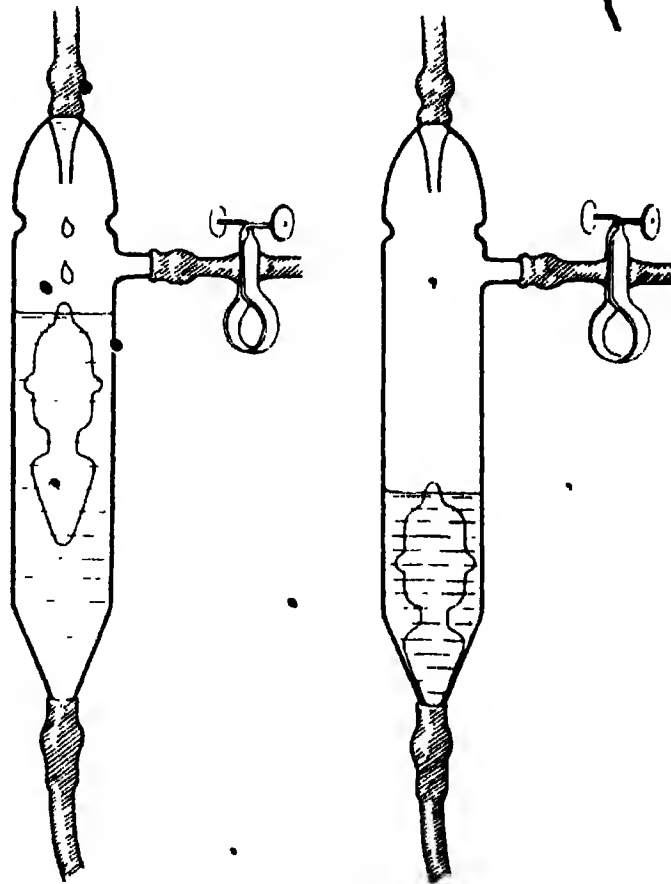


FIG. 4

Safety drip chamber to prevent air embolism.

remembered also that damage to pelvic veins is more liable to be followed by embolism in the Trendelenburg position than when the patient is horizontal. Air embolism has complicated numerous other procedures such as operations on intracranial venous sinuses; irrigation of the maxillary antra and a submaxillary abscess; pneumoperitoneum; Eustachian tube insufflation; filling the bladder with air; æro-urethroscopy in the presence of urethral hæmorrhage; peri-renal insufflation; intra-uterine injections; criminal abortions; vaginal insufflation; tubal patency tests; manual separation of the placenta; Cæsarian section, and even normal delivery at term.

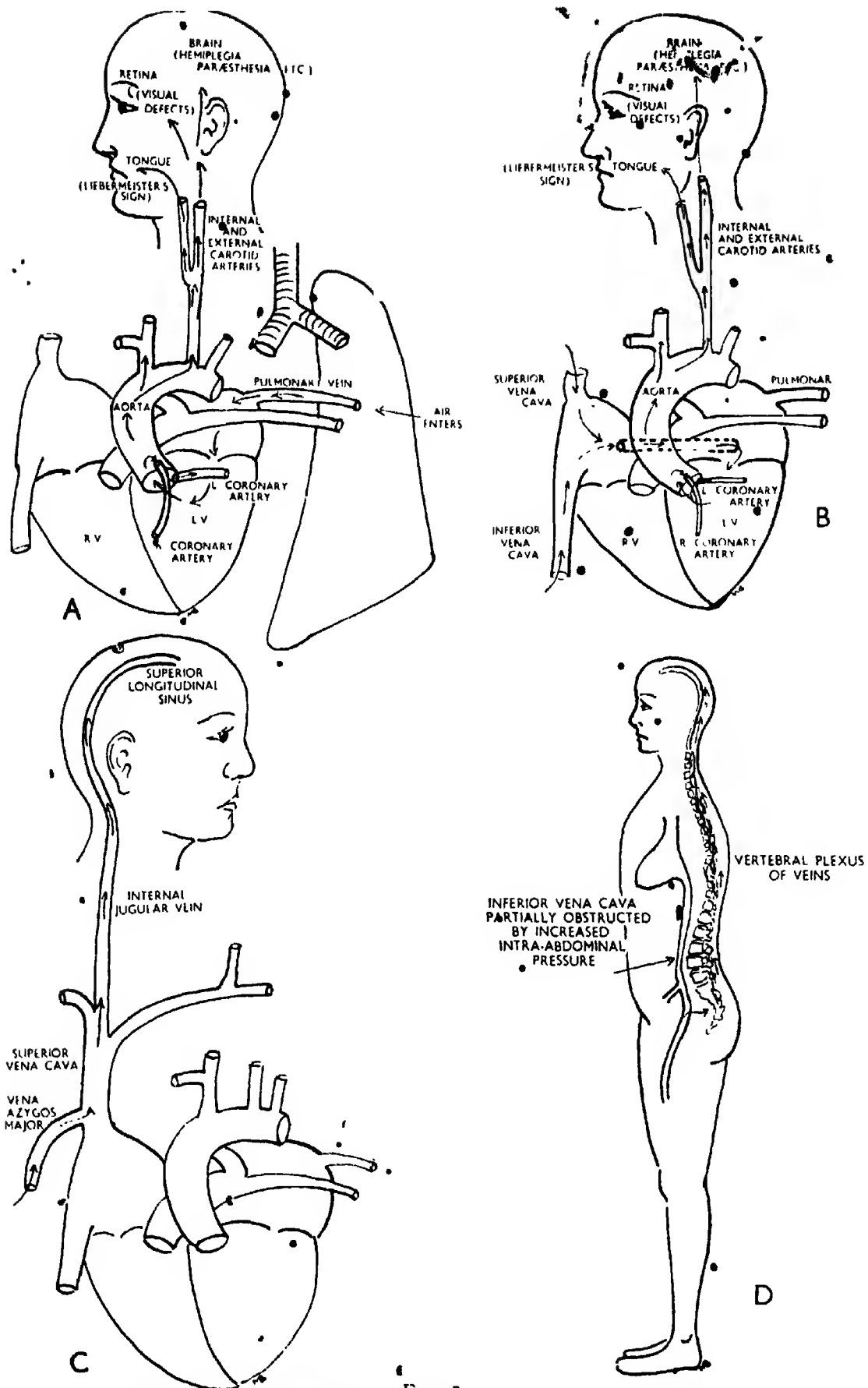


FIG. 5
Showing possible routes by which an air embolus may reach the brain. (The Practitioner.)
[Continued at foot of opposite page]

It may complicate intravenous infusion:—

- (1) If the tubing is loose or cracked, or incompletely emptied of air.
- (2) If the level of fluid in the bottle falls below the orifice of the exit tube. The Barts-Enfield Drip Indicator gives visible or audible warning that the bottle is nearly empty, and so helps to avoid this risk.
- (3) If positive pressure is created in the bottle by a Higginson's syringe when the level of the fluid has fallen below the top of the filter or when the filter is partly blocked.
- (4) The common practice of injecting drugs into the tubing leaves small holes through which air can be drawn particularly if transfusion is being forced by a pump peripheral to the puncture holes. Such injections should be given as near the cannula as possible. The use of translucent tubing by enabling bubbles to be seen in it might obviate this accident.

It is less likely to happen if the clip on the tubing is near the vein rather than near the container, and it may be avoided by using a drip chamber containing a glass float (Fig. 4). This should always be used if a drip is given under pressure. Such a drip chamber must be kept vertical. Arrangement of the tubing to form a loop below the level of the patient's vein is an additional precaution.

When using a vacuum type of bottle to collect blood there is a risk of air embolism in the donor should the vacuum be lost and the tourniquet be released while the needle is still in the donor's vein. This risk could be avoided by inverting the bottle during the collection of blood. Loss of vacuum can be detected by shaking the bottle. A loud clear splash is heard when the bottle

FIG. 5—Contd.

- (A) Air enters a pulmonary venule when a needle punctures the lung and passes via the left auricle and the left ventricle to the aorta.
- (B) "Crossed" or "paradoxical" air embolism. Air enters a systemic venule in the chest wall or elsewhere and passes to the right auricle, and via the patent foramen ovale, to the left auricle, left ventricle and the aorta.
- (C) Air enters a systemic venule and passes via the azygos vein to the superior vena cava where it rises against the blood stream (especially when the patient sits up) and passes up the internal jugular vein to the brain.
- (D) Air enters a systemic venule and passes via anastomoses with the vertebral venous plexus to the superior longitudinal sinus and so by-passes the heart.

contains air at normal pressure, whereas it is difficult to make a vacuum bottle emit even indistinct sounds.

A small amount of air injected at the elbow is known to be harmless, and it is thought that not less than 480 ml. are needed to produce a fatal result, though a much smaller amount might kill an already gravely ill patient. The rate of injection is important. Large amounts of air have failed to kill animals if injected very slowly.

In all serious cases the right heart is distended with frothy blood. There is usually sudden failure of the pulses, and dilatation of the pupils. Auscultation over the heart reveals a characteristic "water-wheel" splashing sound. The patient takes a few deep breaths and is dead within a minute from acute right-sided heart failure.

Recovery has been reported after turning the patient on his left side within forty seconds of his collapse. In this position the air trap is less likely to be in the outflow tract of the right ventricle than when the patient is on his back. The only other measure likely to be beneficial is to aspirate the right heart. The needle is introduced between the fifth and sixth cartilages close to the right side of the sternum and when the ventricle is entered the froth is aspirated. Exposure of the heart may facilitate this.

Arterial air embolism.

The symptoms produced depend on the route taken by the air. Embolism of the coronary arteries can cause death quickly. Syncope, paralysis and paræsthesiæ result from air reaching cerebral arteries. Retinal embolism causes visual defects and air entering peripheral arteries may cause areas of pallor in the skin and tongue.

Apart from intra-arterial transfusion and operations on the left ventricle itself the most obvious route is for air to enter a pulmonary vein, and go via the left heart to the systemic arteries [Fig. 5 (A)]. The accident is most often a sequel to the induction of artificial pneumothorax, but may complicate the opening of a lung abscess and irrigation of an empyema. It does not complicate hæmoptysis. When it occurs during pneumothorax therapy it is caused by the needle entering an adhesion containing a radicle of the pulmonary vein. On inspiration the pressure in these veins is less than atmospheric, and so air is drawn into them. Death can therefore follow the injection of a small quantity of air since

additional air reaches the vein from the lung itself as well as from the needle. Arterial air embolism has complicated submarine escape training from ruptured lung capillaries caused by excessive intrapulmonary air pressure. (Air has also been found in the right heart in these cases, possibly getting there via pulmonary lymphatic vessels and the great veins of the neck). In cases where cerebral air embolism has followed puncture of a systemic vein, "paradoxical" embolism via a patent foramen ovale has been postulated [Fig. 5 (B)].

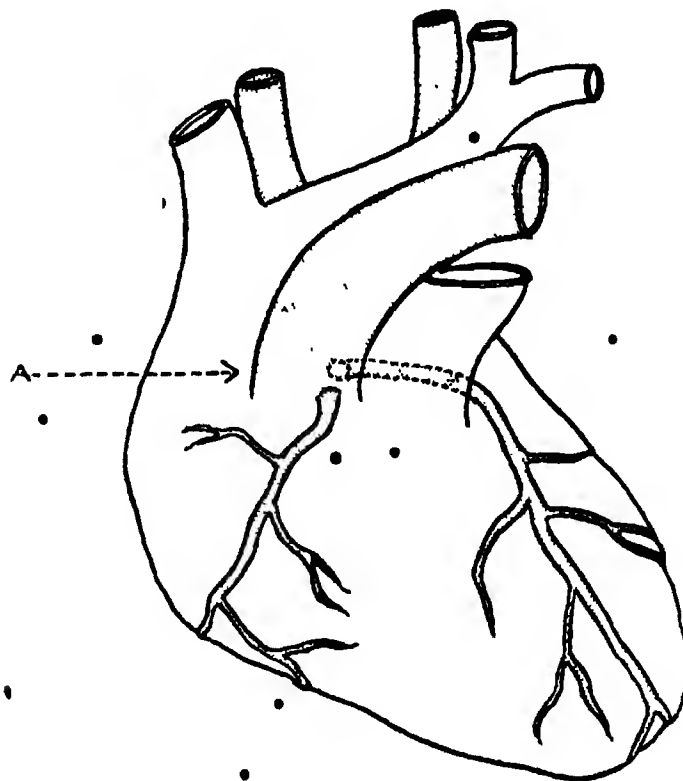


FIG. 6

The heart and great vessels showing the origin of the coronary arteries. It will be seen that a coronary artery does not arise from the part of the aorta marked A (right posterior aortic sinus) which will be uppermost when lying on the left side.

There are two other possible routes, both venous, by which air may reach the brain. Air may enter a tributary of the azygos vein or superior vena cava, and then rise in the jugular vein against the blood stream to the cerebral sinuses [Fig. 5 (c)]. This route may explain those cases of cerebral air embolism in which symptoms do not follow quickly on the insertion of the needle, but come on later, particularly when the patient sits up.

A further route is via anastomoses between systemic (or portal) venules and the vertebral plexus of veins [Fig. 5 (b)]. Air is likely to take this route if for some reason such as coughing or the presence of high intra-abdominal pressure as from pneumoperitoneum, the blood flow in the inferior vena cava is partially obstructed.

Treatment in all these cases consists in immediate cessation of the operation, lowering the head, and putting the patient on his left side. The reason for the left lateral position in arterial air embolism is that it lessens the chance of air entering a coronary artery (see Fig. 6). Even though the patient quickly recovers, it is unwise to resume the operation. Pure oxygen should be given since it washes out nitrogen from the tissues. Morphine should not be given lest depression of the respiratory centre be aggravated.

MISCELLANEOUS MEDICAL ACCIDENTS

Chest aspiration.

If the pump of a Potain's aspirator is wrongly connected it fills the bottle with air under pressure, instead of evacuating it. The air may then be introduced into the chest with immediate collapse of the patient from an acute pneumothorax. Treatment consists in reversing the pump and removing the air quickly (see page 138). Accidents of this type could be avoided if apparatus were made so that it could not be wrongly connected.

Fractured ribs.

Over-vigorous artificial respiration in a frail patient may cause fractured ribs. Coughing may crack a normal rib, though this mishap is more likely if a metastasis is present in it.


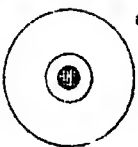



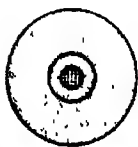

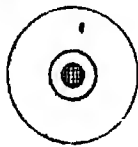
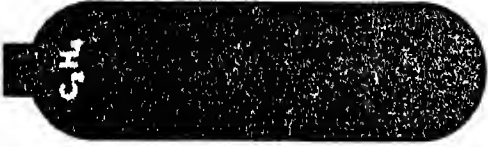






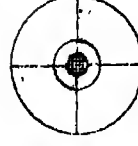


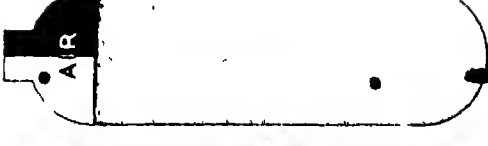

Artificial respiration in the newborn.

Over enthusiastic use of mouth-to-mouth insufflation and of intratracheal oxygen under pressure has caused alveolar rupture and mediastinal emphysema (page 140). The resulting "air block" causes cyanosis and irregular breathing with the chest fully expanded. Diagnosis is confirmed if subcutaneous emphysema appears. After laryngoscopy and gentle tracheal suction, treatment should be on the lines suggested on page 292.

Oxygen therapy in premature infants.

Prolonged administration of high concentrations of oxygen plays a part in causing retrolental fibroplasia in premature infants.

BRITISH STANDARD COLOURS FOR MEDICAL GAS CYLINDERS

	OXYGEN	
	NITROUS OXIDE	
	CYCLOPROPANE	
	CARBON DIOXIDE	
	ETHYLENE	
	HELIUM	
	NITROGEN	
	OXYGEN AND CARBON DIOXIDE MIXTURE	
	OXYGEN AND HELIUM MIXTURE	
	AIR	

If, therefore, ~~the~~ ^{and} ~~vo~~ ^{given} oxygen, it should be in minimum concentration and ~~those~~ ^{minimum} period of time.

Bronchography.

If the crico-thyroid route for bronchography is used it is possible to scratch the laryngeal mucosa and for the iodised oil to set up oedema. Tracheotomy may be required.

If a laryngeal catheter is used and the bronchi have not been previously emptied by tipping the patient, the retained sputum plus iodised oil may be enough to cause respiratory obstruction necessitating abandonment of the operation. Iodism (*see page 43*) is a further hazard. Intubation methods under anaesthesia have been complicated by heart stoppage from vagal stimulation.

The hazards of the cabinet respirator (*page 564*)

In bulbar poliomyelitis, secretions which the patient cannot swallow may be sucked into the lungs with disastrous results. Steep postural drainage (40°) and a sucker should be used in these cases to keep the airway free (*page 565*). Too great negative pressure may cause subcutaneous emphysema. Over-ventilation may cause tetany from alkalosis, but not very easily, because the patient reflexly protects himself by closing the glottis during the inspiratory phase. Acute dilatation of the stomach (*page 467*) is a not infrequent complication and may be caused by oxygen lack affecting the vagus nucleus which leads to atony of the stomach. Atony of the abdominal wall and aerophagy are contributory factors. Adequate pulmonary ventilation and Prostigmin (neostigmine) 0.5 mg. subcutaneously three times a day may obviate this complication. If the box has to be opened in a hurry for some nursing attention the patient may die from anoxia. This can be avoided by the use of a positive pressure respirator while the box is opened as described on *page 566*.

Accidents with gas cylinders (*see also pages 456 and 577*)

Explosion and fire can result from the careless use of gas cylinders. Grit, dust and even minute particles of cotton wool in the outlet of a cylinder may be blown into a regulator valve and cause fire. Cylinders should be turned on and off gently several times before fitting. It is recommended that a cylinder be turned on slightly before connecting to its regulator. Under no circumstances must oil, grease, or grease-containing substances be allowed to come in contact with cylinders or their valves.

Oxygen, as well as mixtures containing cyclopropane, can, in the presence of friction, become explosive. Leaking reservoir or rebreathing bags must be changed immediately.

Accidents have occurred because the wrong gases have been administered. They will be less likely now that the British Standard colours and labels for medical gas cylinders have been adopted (*see plate facing page 33*).

Paracentesis abdominis.

If a Southey's silver tube is used and the guard omitted it is possible for the tube to slip into the abdomen at the time of insertion, or to part from the tubing and slip in later. It is always wise to search the bed clothes first and then to X-ray the abdomen before calling a surgeon.

Suffocation by milk feeds.

- Inhalation of a large vomit may kill an infant. Smaller quantities of inhaled milk cause bronchopneumonia. Small vomits and unskilled and too early feeding of premature babies may lead to fatal post-feeding asphyxia from inhaled milk. These accidents can be avoided by abandoning two common practices, namely, leaving infants to feed themselves from a bottle while lying down, and swaddling tightly after a feed so that movement is prevented.

Similar trouble may follow if a nasal feeding tube enters the larynx. Before giving a nasal feed therefore the position of the tube should be checked. The mouth and nostrils should be gently closed and the proximal end of the tube held under water or close to the doctor's conjunctiva. If the distal end of the tube is in the trachea a current of air will be detected. Polythene tubing, particularly of large diameter (over 0.7 mm.) may perforate the œsophagus in premature infants when used for nasal feeding. Vinyl tubing is more pliable. It would be wise to round the tip or to seal it by heat or wax and make lateral holes in it.

Another risk of careless feeding of infants, and particularly of those with cleft palates, is passage of infected milk up the Eustachian tubes with resulting otitis media.

Incubator hazards.

Faulty insulation in an electrically heated incubator may easily cause fire in the oxygen-enriched atmosphere. Drops of oil and methylated spirit increase the risk. Ether should be avoided for cleaning since it gives rise to formaldehyde in contact with plastic material.

Gastric lavage and vomiting.

The half-comatose patient may bite the doctor's finger (*page 479*). Always, therefore, use a gag or boxwood wedge (*page 540*). Emptying of the stomach may be very hazardous to the comatose patient. Upper oesophageal spasm may allow a tube to enter the larynx or, in the absence of the cough reflex, regurgitated fluid may enter the lungs. An asthma-like reaction with pulmonary oedema then occurs. Gastric lavage in a comatose patient should be performed with the patient prone and with his head over the end of the table (*Fig. 86, page 539*). Alternatively, if an operating table is available, the Trendelenburg position may be used (*Fig. 85, page 538*). We can prove that the tube is in the stomach by showing that fluid which may escape or be aspirated from it after it has been inserted about 18 inches, turns blue litmus paper red. The use of cuffed endo-tracheal and oesophageal tubes are additional safeguards (*page 537*).

The comatose patient is liable to accidents from vomiting and should always be watched until consciousness returns. Should inhalation of stomach contents occur, immediate bronchoscopic aspiration is desirable. It is of little use once pulmonary oedema has developed (*page 468*). In an infant, an attempt may be made to aspirate milk from the bronchi by a large rubber catheter introduced through a laryngoscope. Failing these measures, postural drainage should be adopted. It would be wise to start penicillin treatment (*page 601*) at once.

Ruptured mercury bougie.

The swallowing or aspiration of mercury from this accident creates a feeling of emergency but the situation is not dangerous. Metallic mercury is innocuous when surrounded by intact epithelium. Postural drainage should be used to aid its clearance from the lungs.

Swallowed Ryle's tube.

When a Ryle's tube is completely swallowed by accident don't convert the situation into an emergency by operating too soon. If there are no ill effects wait a long time (several weeks) before operating. Always use X-rays *immediately before* operation for the tube may have passed on.

Electric convulsion therapy (Electroplexy).

Temporary slowing of the heart and cyanosis are commonly encountered after electrically induced convulsions. Occasionally

the heart may stop and an intracardial injection of adrenaline or 5 ml. of 10 per cent. calcium chloride (*page 44*) may be needed (*page 20*). Failing this, mechanical stimulation by a blow on the chest may be used. Artificial respiration may be necessary to restart breathing after convulsions. Very rarely status epilepticus has occurred and thiopentone has had to be given intravenously. Surgical complications, namely fractures, may also be present but can be avoided by preliminary use of a muscle relaxant.

Gastroscopy.

There is slight but definite risk (6.5 per 10,000 cases) of tearing the œsophagus during gastroscopy, especially in elderly kyphotic vitamin-starved women. Most cases of œsophageal perforation have not been associated with difficulty of passage. With the modern flexible instrument the tear occurs in the region of the post-cricoid sphincter where the outer longitudinal muscle fibres of the œsophagus are deficient, and the tip of the instrument is apt to be held up by poor swallowing or spasm. Extension of the head at the outset or later, in an attempt to increase the field of view adds to the risk. An unduly prolonged examination (over 10 to 15 minutes) carries the risk of causing pressure necrosis of the œsophagus.

The symptoms of œsophageal rupture are dysphagia and substernal pain followed by surgical emphysema of the neck. When emphysema is absent (an X-ray examination may be necessary to exclude it) the other symptoms may be due to œsophagitis. In either case the treatment is to give:—

- (1) Penicillin lozenges (1,000 units in each), one every half hour as far as possible by night and day.
- (2) Penicillin or other antibiotic by intramuscular injection (*page 601*).

The mouth should be moistened, but swallowing of fluids avoided for 24 hours, and the fluid requirements (*page 463*) made up by rectal or intravenous drip. As suture of the tear has been done successfully the advice of a surgeon should be sought.

Bronchoscopy.

The hazards of bronchoscopy include:—

- (1) THOSE DUE TO THE ANÆSTHETIC.—Local anæsthetics, particularly cocaine, produce their own emergencies (*page 23*). The use of ether carries the potential risk of explosion. Thiopen-

tone (Pentothal), if employed as the sole anæsthetic agent, is especially dangerous because after its use, any stimulus to the larynx may set up intense spasm. This is a very serious emergency since it quickly leads to asphyxia and prevents the passage of the bronchoscope through which oxygen might be given. Artificial respiration is, of course, useless. Relaxation of the cords may occur even when death seems imminent but it should not be awaited since it can be produced by the prompt intravenous injection of 20 mg. of succinylcholine. When relaxation occurs, the bronchoscope should be slipped through and oxygen blown in at 8 litres a minute. Artificial respiration might also be used at this stage to make recovery doubly sure. Failing all else tracheotomy (page 569) should be performed and will give instant relief.

(2) TRAUMA.—Damage to the teeth and hyoid bone, and perforation of the wall of the respiratory tract, are all possible, but unlikely. The risk of oral damage can be lessened by the use of a light plastic shield fitting over the teeth. Passage of the bronchoscope through a tuberculous larynx may set up acute œdema, and for this reason bronchoscopy is best avoided in patients with laryngeal tuberculosis. If it must be done, a tracheotomy set should also be at hand. Similar trouble may complicate the removal of tuberculous granulation tissue from the larynx.

(3) BLEEDING.—Taking a specimen for biopsy, particularly in the case of an adenoma, may lead to profuse bleeding necessitating aspiration of blood through the bronchoscope. In one case of multiple bronchial papillomata, bleeding was stopped by inducing artificial pneumothorax. A blood transfusion may be needed.

Retained blood may lead to massive collapse. To prevent blood from passing into the lung, drainage should be promoted by lowering the head. The return to consciousness can be hastened by giving Injection of Leptazol B.P. 2 ml. intramuscularly, and the cough reflex is made more sensitive by injecting strychnine hydrochloride 7.5 mg. (gr. $\frac{1}{8}$) intravenously.

In bronchoscoping tuberculous patients the healthy side should be examined first to avoid the risk of carrying over tuberculous sputum. The risk of laryngeal spasm and œdema in a child and the poor view through a small bronchoscope should make us avoid bronchoscopy under the age of 3 years except for very definite indications.

Cardiac catheterisation.

The main hazards are cardiac arrhythmias and thrombosis of the vein at the site of insertion of the catheter. Air embolism is unusual. Less serious mishaps are venospasm and syncope. If a chemically sterilised catheter is introduced as far as a branch of the pulmonary artery infarction may follow. It would be safer to use an autoclaved catheter.

Contraindications are ventricular tachycardia, recent pulmonary or cardiac infarction and bacterial endocarditis. The mortality rate is less than 0.1 per cent. Cardiac catheterisation should only be done by those with special experience in the method. Any unusual feeling in the patient or persisting abnormality of rhythm should lead to termination of the examination.

Angiocardiography.

The chief hazard is syncope from sensitivity to diodone especially in patients with congenital heart disease. It cannot be predicted by preliminary testing or prevented by premedication.

EMERGENCIES FOLLOWING INJECTIONS**Cracks in ampoules.**

" Invisible cracks " in ampoules may lead to contamination of their contents with the antiseptic fluid in which they are stored. This should, therefore, have a distinctive colour or, preferably, the ampoule should be stored dry to avoid all risk of contamination. Even so water may be selectively lost from such an ampoule and lead to concentration of its contents. The practice of inverting a rubber-capped bottle in antiseptic should be similarly condemned as negative pressure may cause antiseptic to enter.

Accidental intravenous injection.

The plunger should always be withdrawn a little after inserting the needle subcutaneously or intramuscularly and before injecting fluid to make sure that a vein has not been entered. When this cannot be done as in mass inoculations where only the needle is sterilised and contamination of the syringe must be avoided the needle should be inserted into a pinched-up fold of skin which is very unlikely to contain a vein. In deeper injections if the needle can be turned through a right angle it is unlikely to be in a vein.

Accidental injection of a toxic dose.

It may happen that an overdose of morphine or other drug is injected inadvertently. If this is subcutaneous or intramuscular prompt action may obviate ill effects. A tourniquet should be applied at once to prevent absorption and released at suitable intervals, so that a toxic dose is not absorbed and gangrene is avoided. Should the dose be very large, it might be advisable to incise the part and irrigate it with saline. If application of a tourniquet is impracticable, absorption can be delayed by application of ice or subcutaneous injection of adrenaline. In the case of morphine the antidote nalorphine (Lethidrone) (*see page 14*) should be given. Adrenaline overdosage (minimum lethal dose 4 mg. = 4 ml. of 1 in 1,000 solution; maximum tolerated dose 8 mg.) calls for the use of an adrenergic blocking agent such as phentolamine (Rogifine) (*see page 258*).

Allergic reactions.

SKIN TESTING.—Intradermal injection of skin testing material is occasionally followed by alarming reactions characterised by severe local swelling, generalised urticaria and asthma. Overdosage from careless reading of the label may be responsible. Skin testing, particularly with animal hair and dandruff, should be avoided in children and certainly if there is a strong history of allergy. Death has occurred from multiple skin testing in a very sensitive subject.

A tourniquet should be applied to the limb—indeed it is wise to place a sphygmomanometer cuff in position proximal to the site of injection before starting. Adrenaline, etc., should be used as for anaphylaxis.

THERAPEUTIC INJECTIONS OF ALLERGENS.—An overdose of allergen or a therapeutic dose in a very sensitive person may cause similar reactions to those described above, and should be treated similarly. Non-protein substances such as intravenous sclerotics or any organic compound may be responsible. Inadvertent injection of material into a vein will cause an immediate constitutional reaction, but no local swelling. Damage to a vein during injection may allow material to enter the blood stream after an interval and cause a severe “back seepage” reaction. In such cases there is an ecchymosis at the puncture site.

Treatment.—The site of the allergen injection should be pressed on to limit absorption and adrenaline, etc. given as for anaphylaxis.

VACCINES.—Anaphylactic reactions may follow injection of vaccines prepared from chick embryos (influenza, typhus and yellow fever) in those who are allergic to egg.

Anaphylactic shock.

Though rare this is always an alarming accident. It comes on during the intravenous injection or up to two hours after the intramuscular injection of serum in an allergic patient or in one who has had serum of similar origin more than ten days previously. The earlier the reaction the more dangerous it is. It may follow injection of substances other than serum to which the patient is sensitive.

The earliest symptoms are sweating, pruritus, cough and wheezing. Then a feeling of constriction in the chest and abdominal pain with vomiting are experienced. The patient is obviously very ill and acute circulatory failure, coma and death may quickly follow.

Prevention.—The accident can be avoided by giving a small trial dose and seeing what happens in the next half hour (page 579). Any reaction to this dose will be mild and easily treated. This method is more reliable than preliminary skin testing which, in any case, only shows local rather than general sensitivity.

Treatment.—Adrenaline, aminophylline and intravenous antihistamines should always be at hand when serum is being given (page 579). Give 0.3 to 0.6 ml. (or 5 to 10) adrenaline 1 in 1,000 subcutaneously and repeat in ten minutes if necessary. Apply a tourniquet above the injection site. If the response is poor give an aminophylline preparation by injection (page 598). Should this fail to cause improvement promezathine hydrochloride (Phenergan) 25 to 50 mg. should be given intravenously (page 610).

Poliomyelitis following injections.

Immunising injections may be followed by poliomyelitis. The present evidence suggests that it would be wise not to inject diphtheria and whooping cough prophylactics, and particularly alum precipitated prophylactics and mixed ones, during an epidemic of poliomyelitis and to avoid especially children over the age of one year.

Neurological sequelae of prophylactic inoculations.

Complications at all levels of the nervous system may follow the injection of sera and various prophylactics. They are quite

rare and not usually of an urgent nature. As they probably result from anaphylactic hypersensitivity treatment by antihistamine drugs would seem wise.

Vaccination.

In sufferers from chronic eczema vaccination may cause severe generalised vaccinia with a mortality as great as that of smallpox.

Post-vaccinial encephalomyelitis (*see also page 318*) is a rare complication. It is mostly confined to primary vaccination of adults and can be minimised by primary vaccination in infancy.

Vaccination may involve the risk of contracting poliomyelitis during an epidemic and should be postponed except when an outbreak of smallpox occurs simultaneously.

EMERGENCIES FOLLOWING THE USE OF CERTAIN DRUGS

The Jarisch-Herxheimer reaction.

This rare upset follows within twelve hours of the first injection of penicillin usually in a case of neuro-syphilis and less often in cardio-vascular syphilis. It may be local and is then thought to be due to oedema following massive destruction of spirochaetes and liberation of toxin in a vital organ, such as the brain or heart. The rapid softening of the wall of an aneurysm, acute oedema in a gummatous larynx, convulsions and coma in G.P.I. and paraplegia in syphilitic lesions of the cord may all result.

In pre-clinical and early syphilis, a general reaction results in headache, malaise and fever. If these symptoms follow the injection of penicillin for gonorrhoea, the co-existence of syphilis should be suspected. Sometimes a syphilitic rash is brought out or accentuated. Pyrexia to 100°F. is very common following penicillin for neurosyphilis. The intensity of the reaction does not appear to be related to the dose of the spirochaeticidal drug but very gentle treatment (20,000 units of penicillin daily) in the early stages is advisable.

If a reaction occurs the treatment should be symptomatic. Anti-histamine drugs have not been found helpful.

Excretion, Urography.

General reactions (vomiting, flushing and sensations of heat) may occur with any of the drugs commonly employed for excretion urography. This suggests a common cause but it is generally

agreed that this is not iodine. Reactions may be delayed in patients with poor renal function and excretion urography should be avoided in such cases.

There does not seem to be any reliable test of sensitivity and deaths have occurred in cases with negative tests. But it would be wise, for medico-legal reasons, to use one particularly if there is an allergic history. An intradermal injection of 0.05 ml. of the drug may be made. Local swelling indicates sensitivity. If the history or the test is positive and urography is essential give 1 ml. of a 1 in 100 dilution of the strength usually employed and at ten minute intervals 1 ml. of 1 in 10 dilution and 1 ml. and then 5 ml. of full strength solution. If no reaction follows, the full dose may be given. A previous reaction calls for desensitisation by even slower steps. In all cases a pause of 30 seconds after the injection of the first 2 ml. is wise.

Should an immediate reaction occur the injection should be stopped and adrenaline given. A reaction with iodoxyl does not necessarily indicate sensitivity to diodone but in subsequent investigations the retrograde method using sodium bromide would be preferable.

Retrograde pyelography.

Fatal bromide poisoning followed the inadvertent retention in the bladder of 10 fl. oz. of 25 per cent. sodium bromide. The mucosa was inflamed and so absorbed rapidly. The bladder should always be emptied after pyelography and preferably a less toxic contrast medium (*e.g.* diodone) should be used.

Mercurial diuretics.

Sudden death may occasionally follow immediately on the intravenous (but not the intramuscular) injection of a mercurial diuretic (Mersalyl B.P.) (*page 654*). In most cases previous injections have caused no ill effect and this suggests that acquired sensitivity may play a part. The blood proteins have usually been low, from nephritis.

It is best to avoid intravenous mercurials in all cases other than those of congestive cardiac failure. If, however, the intravenous route is used the addition of 0.5 ml. of 20 per cent. magnesium sulphate solution is said to make it safer. Injections should not be given more frequently than once in three days. Should an accident happen and if there is time Injection of Nikethamide B.P. 2 ml. should be given intramuscularly and an

intracardiac injection of adrenaline made (see page 18). It would be reasonable in a case not immediately fatal to give 300 mg. of BAL (Dimercaprol) intramuscularly.

A brisk mercurial diuresis may precipitate an attack of gout in a gouty subject (see page 485). In the elderly mercurial diuretics may precipitate uræmia by inducing sodium loss, particularly if the salt intake is low.

Paraldehyde.

This has caused serious inflammation when given by rectum or by mouth because it has been slowly converted into acetic acid. Paraldehyde bottles should be of coloured glass and kept in a dark cool place. It is well to check them periodically.

Anti-thyroid drugs (Carbimazole. Thiouracil).

On rare occasions, these cause agranulocytosis. Routine white cell counts are, however, useless in its early detection, since the typical disappearance of polymorphs may be very rapid in onset. If a patient taking these drugs develops pyrexia and a sore throat, he should be given penicillin 500,000 units at once and a white cell count made. Should agranulocytosis be found, continue with penicillin and also give pentnucleotide and pyridoxine (see page 177).

Enteric coated tablets.

If these are, mistakenly, crushed to facilitate administration to a child trouble may result. Hexylresorcinol (for thread worms) so administered has damaged the mouth.

Contaminated eye drops.

Watery fluorescein eye drops have been found to be easily contaminated with *Pseudomonas pyocyanea* which will cause serious damage when there is a corneal abrasion. Only drops made up with Solution for Eye Drops B.P.C. and N.F. are safe. Alternatively Lamellae of Fluorescein 0.26 mg. (gr. $\frac{1}{250}$) should be used (page 2).

Iodism.

Very rarely acute iodism follows injection of iodised oil into the bronchial tree. Tests for iodine sensitivity are not very reliable but at any rate will show that "reasonable care" was taken. Weak Solution of Iodine B.P. 0.3 ml. (m 5) in a little milk should

be taken on the previous day and the patient watched for a rash or coryza. The newer preparation for bronchography, propyliodone (Dionosil Oily) is less likely to cause trouble.

If iodism occurs it is only rarely dangerous though it may cause alarm. Treatment depends on facilitating the excretion of iodine compounds in the urine. Simply stopping administration of the drug and insisting on the patient's drinking freely—about eight pints of fluid a day—usually suffices. Calamine lotion should be applied to the eruption; bullæ should be snipped with sterile scissors and penicillin given (*see page 601*). In an iodine sensitive patient brominized oil (May & Baker) could be used for any subsequent examinations.

Dangerous combinations of drugs.

Sometimes drugs innocuous in themselves prove dangerous in combination. Parenteral injection of soluble calcium salts potentiates the action of digitalis glucosides and increases the irritability of heart muscle. Occasionally, a digitalised patient has died from ventricular fibrillation induced by calcium given intravenously. It is recommended that digitalis should be stopped for four days before calcium is given intravenously. In all cases, intravenous injections of calcium, and particularly of calcium chloride, should be made very slowly (*page 19*). Calcium is, therefore, not as suitable as other substances such as saccharin for determination of the circulation time (*see page 127*). Another dangerous combination is adrenaline during chloroform and trilene anæsthesia. The effects of morphine, barbiturates, bromides and alcohol are at least additive and may be synergistic. Before administering one of these drugs the doctor should always consider whether the patient is under the influence of another.

OTHER REACTIONS FOLLOWING INJECTIONS

Numerous examples could be found of emergencies due to injection of the wrong drug. The label should always be read carefully because some drugs such as carbachol are put up in various strengths of which only the weaker ones are suitable for injection. If the label is missing or illegible the drug should not be used. Even as innocuous a substance as distilled water has caused trouble from hæmolysis when given intravenously.

Emergencies may also arise from the injection of the right drug by the wrong route, Adrenaline, unless very dilute and

given very slowly, may cause death from ventricular fibrillation if injected intravenously. Less serious results are severe blanching, headache, palpitation, vertigo and occasionally hemiplegia from vascular spasm. As adrenaline is rapidly oxidised in the tissues, useful therapeutic intervention is impracticable, but should adrenaline 1 in 100, which is meant for aerosol use, be injected it would be wise to counteract its effect by inhalation of amyl nitrite (*see also page 39*). Carbachol given intravenously may cause collapse. The clinical picture of fat embolism (*page 135*) may result if oily solutions are injected into veins. Aspiration should therefore always be tried before giving an intramuscular injection particularly of an oily solution. Too rapid intravenous injection of many solutions, especially cold ones, will cause chest pain and rigors. Ampoules of cold solutions should be warmed in hot water (110°F.) before use, or the syringe containing the drug should be "sealed" with a hypodermic needle and placed in hot water. Inadvertent injection of a vaccine into a small vein may quickly produce an alarming result. Material contaminated with bacteria or their products will cause sharp febrile reactions if given intravenously. If symptoms do not abate in a few hours, blood should be taken for culture and penicillin therapy started. If a patient complains of a queer taste in the mouth after a subcutaneous or intramuscular injection one should suspect that the material has entered a vein and particularly if there is some bleeding at the puncture site.

Soluble sulphonamides cause disastrous results if used intrathecally and should never be given by this route. Care must be taken not to use intrathecally penicillin to which phenol has been added as a preservative since, though innocuous elsewhere, it may cause a severe and even fatal reaction. Procaine Benzylpenicillin is not simply a mixture of penicillin and procaine but a new compound which can cause severe reactions if injected intravenously. Even when injected intramuscularly it may cause anaphylaxis and so anti-histamines should always be at hand when procaine penicillin preparations are used.

BLOOD TRANSFUSION REACTIONS

These may be immediate or delayed.

IMMEDIATE REACTIONS.

When something goes wrong very soon after a blood transfusion is started there are three main groups of conditions which may be responsible.

- (1) Non-specific factors connected with the apparatus and fluids used.
- (2) True blood transfusion reactions due to intravascular hæmolysis.
- (3) Circulatory overloading.

In addition the risk of transmitting disease may constitute an emergency.

Non-specific reactions.

These include pyrexia, chills and rigors. They result from too rapid transfusion or the presence of foreign protein often of bacterial origin in the transfused fluid. New rubber, especially synthetic, and tubing from which chalk has not been removed by boiling in 0·1 per cent. caustic soda may be responsible for rigors. Hæmolysed blood and blood which has been allowed to warm up may also be responsible. Once blood has left the refrigerator it should be used without delay and if unused must be discarded. Intense pain in the back, which is so characteristic of intravascular hæmolysis, does not occur.

Treatment.—In the absence of evidence of a true blood transfusion reaction it is usually only necessary to slow down the rate of transfusion. If this is ineffective, the source of the blood and of the anti-coagulants added should be scrutinised, and if they are suspect a new bottle of blood substituted.

Exchange transfusion carries the additional risk of lowered ionised calcium causing tetany and cardiac depression because of citrate overdosage. The special preparation of blood to avoid this is cumbersome and if citrate intoxication is suspected it is probably best treated by small doses of 10 per cent. calcium gluconate cautiously administered. The risk of raising the plasma potassium too high is obviated by using fresh blood.

True transfusion reactions.

These are rare and due to technical (*e.g.*, faulty grouping) or clerical errors. They appear usually before 100 ml. of blood have been given. Reactions will occasionally occur despite any demonstrable incompatibility. Transfusion always therefore carries a risk and should only be carried out when clearly indicated. In injured patients because they may have renal anoxia, in dehydrated

patients and in women near full term, particularly if there is pregnancy toxæmia, any transfusion reaction, whether from incompatibility or from old blood, is apt to be severe.

There are two clinical types of reaction. The first is sudden circulatory collapse or shock and carries a high mortality. The second type is somewhat less serious. The patient complains of very intense pain in the back and tightness in the chest. He has a rigor and becomes cold, clammy and cyanosed. This reaction may be masked by anæsthesia and so, when a blood bottle is changed during an operation, the blood pressure and pulse rate should be watched carefully for ten minutes or so afterwards.

In a severe case there follows complete anuria but milder reactions may be missed because some urine is passed. Every transfused patient should be put on a fluid output chart for 48 hours. A low output of urine of specific gravity under 1.010 indicates renal damage.

As in all procedures there is need for common sense. For example, an "A" patient who receives many pints of "O" blood may have a reaction if he is given "A" blood shortly afterwards. It is unwise to give even group "O" Rh. negative blood indiscriminately without expert cross matching and the old idea that the provider of such blood was a "universal donor" is dangerous. Even blood which is satisfactory to ordinary cross matching is not free from danger because the recipient may possess antibodies to one of the other blood group systems, particularly if previously transfused.

The history of the outcome of pregnancies and previous transfusions should always be obtained and if this suggests that the patient may be a "dangerous recipient" then plasma or a plasma substitute should be given pending expert cross matching with the employment of the indirect Coombs' test. Blood for cross matching should be taken *before* giving a plasma substitute as this may interfere with the test. Blood grouping should always be done by an expert, if possible, but in a dire emergency one might have to be satisfied with simple cross matching alone (*see page 588*).

Treatment.—A sudden reaction means immediate cessation of the transfusion. Circulatory collapse should be dealt with as described on *page 152*. If pain in the back occurs compatibility tests should be repeated and compatible blood given. The object is to keep up the blood pressure and prevent renal failure. Should this develop the patient is unable to excrete water, metabolites and

electrolytes. He should be treated as described under renal failure (page 262).

Circulatory overloading. (*For risks in children see page 280*)

In a previously healthy patient rendered rapidly anæmic and shocked by blood loss, the danger of circulatory overloading is slight. Indeed rapid transfusion is desirable to restore the blood pressure to normal. Up to 70 per cent. of large losses of blood can be rapidly replaced but the transfusion should be stopped if the jugular veins become distended. In the chronically anæmic patient with feeble heart muscle, the danger of a too rapid transfusion of blood is very real. Should such a person's anæmia call for transfusion a concentrate of cells should be given by first removing the plasma by aspiration ("packed cell" transfusion). When time for blood dilution after hæmorrhage has occurred the packed cell volume (average normal about 45 per cent.) is a useful guide to the amount of packed cells needed, since the cells from 500 ml. of blood will raise the packed cell volume by 5 per cent.

Starling's work showed that a rise in venous pressure is associated with increased cardiac output; within certain limits, the healthy heart adjusts itself to deal with as much blood as may be poured into the right auricle. A stage may be reached, however, at which this physiological mechanism breaks down; it occurs much earlier in patients suffering from anæmia than in healthy subjects. It is apt to supervene with dramatic suddenness during a too rapid transfusion of a patient suffering from long-standing anæmia. The first symptom is a persistent cough and this is a signal to stop the transfusion. Frothy sputum, sometimes blood stained, next appears and the patient becomes dyspnoëic and cyanosed. Auscultation reveals moist sounds. This picture is liable to appear if the rate of transfusion exceeds 1 ml. per lb. per hour, *i.e.* 140 ml. per hour for a 10 stone man (or half this rate if the hæmoglobin level is 25 per cent. or less). In a less acute form the water-logged lungs become the seat of pneumonia.

The average drip bulb kept at one drop per second should deliver 3 ml. per minute or 180 ml. per hour since the size of the average drop is $\frac{1}{20}$ ml. The actual amount delivered will be less, for the drip slows as the head of pressure falls. Resistance from venous spasm will also slow the drip. A useful rule for infants is that the normal requirement of 142 ml. per Kg. ($2\frac{1}{2}$ fl. oz. per

lb.) per 24 hours will be achieved if the number of drops per minute equals the weight in pounds (*see also page 281*).

Treatment of circulatory overloading consists in:—

- (1) Stopping the transfusion.
- (2) Giving morphine 15 mg. (gr. $\frac{1}{4}$) and atropine 1 mg. (gr. $\frac{1}{60}$) subcutaneously.
- (3) Venesection. Remove half to one pint of blood, according to the severity of the symptoms and the volume of blood transfused. Alternatively tourniquets can be applied to all four limbs.
- (4) Administration of oxygen.

TRANSMITTED DISEASE.

The risk of transmitting malaria and syphilis from the donor to the recipient is slight if healthy donors only are used, and those who have ever suffered from malaria or who have positive Wassermann or Kahn reactions are rejected. A week's refrigeration eliminates the danger of syphilis. A greater risk is that of transmitting infective hepatitis since the virus cannot be detected in the donor. If there is a history of jaundice in the preceding two years the donor should be rejected. In none of these cases can the situation be classed as an emergency.

The risk of inducing a virulent septicæmia from contamination of stored blood exists, and immediate symptoms would result. Accidental reversal of the syringe might transfer infection from the recipient to the donor in the now rarely-used direct arm-to-arm transfusion. Prompt treatment by antibiotics would be indicated in each case.

DELAYED REACTIONS.

In blood transfusions our chief concern is that the donor's cells (containing agglutinogens) shall not be agglutinated by the recipient's serum (containing agglutinins).

If a group "O" donor gives blood to a recipient who is not group "O" the donor's plasma may agglutinate and hæmolyse some of the recipient's cells and cause delayed jaundice. This, however, is not serious, because the donor's plasma is small in amount and its agglutinins of low titre. Also, all the cells of the recipient's tissues, and not only his red blood corpuscles, absorb the transfused agglutinin.

Similarly patients who belong to the 15 per cent. whose red cells do not contain Rh. agglutinogens (*i.e.*, who are Rh. negative) will in two-thirds of cases be sensitised by a transfusion of Rh. positive blood and a subsequent transfusion will cause the production of antibodies. Pregnancy with a rhesus positive foetus will also cause sensitisation.

Since 85 per cent. of blood is Rh. positive, the risk is considerable, and blood selected by the ordinary "A,B,O" compatibility tests is very likely to be incompatible in Rh. negative patients. These risks do not apply to Rh. positive recipients since anti-Rh. agglutinins do not occur naturally and only appear as a result of transfusion or pregnancy. Rh. negative blood should therefore be used to transfuse Rh. negative patients, particularly for second transfusions, and for pregnant women who have been previously transfused.

Group "O" does not simply mean that "A" and "B" agglutinogens are absent. It implies the presence of a specific "O" blood group substance but as this is a very poor antigenic stimulus repeated transfusions of "O" blood rarely give rise to anti-"O" agglutinins. There are other blood groups, however, such as Kell (10 per cent. of population) and Duffy (65 per cent. of population). If a Kell negative recipient is transfused from a Kell positive donor he has a greater chance of a hæmolytic reaction if transfused again from the same donor. This can be prevented by cross matching but if the donor were a husband and the recipient his young wife one transfusion could sensitize her so that her subsequent children would be injured. For this reason it is unwise to transfuse a young wife with her husband's blood.

True transfusion reactions should be reported to the blood supply depot concerned. A few ml. of blood should be left in the bottle and kept for 24 hours after every transfusion lest a reaction should occur and investigation be wanted. Any pre-transfusion specimen of the recipient's blood should also be kept.

Transfusions and even intra-muscular injections of Rh. positive whole blood into an Rh. negative infant (*see page 298*) may cause Rh. antibodies to develop and expose the child to transfusion risks in later life, and also, in the case of a girl, to the risk of having still-born infants or ones affected by hæmolytic disease of the newborn. To avoid these delayed risks we should give only

Rh. negative blood to all female children and fertile women of child-bearing age who are un-grouped or Rh. negative.

• **Hazards to the donor.**

Even the donor is not immune to certain hazards such as fainting attacks and the risks inherent in needling.

IATROGENIC EMERGENCIES

It should not be forgotten that almost all the procedures we use in our daily work, even the most trivial of them may, under certain circumstances, cause emergency situations. This applies not only to the technical procedures already discussed but also to the routine methods of examination. We should not alarm our patient, for example, by raising our eyebrows on the unexpected discovery of serious disease or by thinking aloud.

Careless use of words has frequently caused collapse of patient and relatives. "Watch your word" is a good maxim for the doctor faced with an emergency, for his word is a therapeutic instrument no less powerful to avert and no less dangerous to produce an emergency than a surgeon's scalpel.

C. ALLAN BIRCH.

CHAPTER IV

Acute (Non-Surgical) Abdominal Catastrophes

THE four main features of an abdominal emergency are pain, rigidity, vomiting and distension. All of these have well recognised "surgical" causes, but each may be produced by a medical condition and mislead the surgeon into undertaking an unnecessary laparotomy. A helpful way of dealing with the subject is to consider each symptom or sign in turn, noting what medical condition may cause it and how it may be distinguished from the same symptom or sign attributable to a "surgical" cause. Each condition will have to be considered in some detail, since the physician's problem is not that of the surgeon, namely, whether to operate or not. While in most of the conditions which follow, acute abdominal pain is present, it is rarely persistent. *The old rule still holds that the majority of abdominal pains lasting longer than six hours are caused by conditions of surgical import* (Zachary Cope). If there is real doubt it is best to operate for it is probable that more harm is done by remembering the medical snags than by forgetting them.

It is important to be on guard against the presence of an acute medical and surgical condition at the same time. Thus pyelitis and pneumonia may occur with appendicitis, and coronary occlusion with cholecystitis. Another source of trouble is the patient who is known to have had coronary thrombosis and who later has another somewhat similar attack which turns out to be a perforation.

Errors constantly occur because the surgeon neglects to use the stethoscope, or the physician the finger stall. No method of bedside examination is the prerogative of any one kind of doctor. Remember how Osler defined a consultant—"a man who makes the rectal examination after the other physicians passed it up."

The medical "acute abdomen" is not a rarity, and has been found to account for 15 per cent. of cases presenting abdominal symptoms. More than one of the cardinal signs are usually present

but one is outstanding. In this account we shall be concerned almost entirely with differential diagnosis.

PAIN

Abdominal pain causes most of our difficulties and is of two main types, visceral and peritoneal, according to its origin.

Visceral pain is caused by distension or spasm of a viscus. It is diffuse; it comes and goes and is unassociated with rigidity or pyrexia. Tenderness is minimal. Often the patient is restless. Biliary colic is a good example, while of the extra-abdominal causes, coronary thrombosis produces many of the features.

Peritoneal pain is caused by irritation of the peritoneum. It is localised, constant, and associated with rigidity and often with pyrexia. Tenderness is marked and the patient lies still. Perforated gastric ulcer causes this type of pain which also occurs as a reflex phenomenon in pleurisy.

In addition to pain, of which the patient complains spontaneously, we must also mention tenderness, or pain elicited on palpation. A useful point in the differentiation of tenderness caused by some extra-abdominal condition is that in true peritonitis there is definite tenderness of the pelvic peritoneum on rectal or vaginal examination.

Tenderness is very important in children and if a conscious child makes no attempt to remove the palpating hand, the condition is probably not a local one such as appendicitis.

The fact that severe abdominal pain is uninfluenced by ordinary doses of morphine may indicate the presence of a surgical condition demanding operation—except for two conditions, tabes and pancreatitis, for which operation is not needed. But there is no easy rule and all depends on the history and physical signs interpreted by the judgment, knowledge and experience of the surgeon.

Coronary thrombosis.

Coronary thrombosis with abdominal pain is a constant source of difficulty in its differentiation from a perforated ulcer. A history suggestive of coronary disease is more helpful than one of indigestion, particularly if it can be established that there has been pain brought on by exertion, and if the present acute pain began behind the sternum rather than in the abdomen. A

clear history is, however, often unobtainable and we are faced with a patient who has sudden severe overwhelming epigastric and retrosternal pain. Usually by the time we see him, radiation has occurred. If this is up into the chest and down the arms it is in favour of coronary thrombosis, while radiation over the abdomen favours perforation. Shoulder and scapular pain especially on the right side is common after perforated duodenal ulcer with a subdiaphragmatic collection of fluid.

Other factors help us to decide. The pulse in perforation is slow at the onset, whereas it is rapid in coronary thrombosis. Respiration is shallow and thoracic in perforation, but abdominal in coronary thrombosis. Rigidity in perforation, other than into the lesser sac, is extreme and persistent, whereas the rigidity caused by cardiac infarction is not board-like and varies with respiration. Circulatory failure from the shock of perforation soon passes off, but in coronary occlusion it steadily dominates the picture. In perforation, escaped gas causes the liver dullness to disappear. As distended bowel may prove confusing by also obscuring the normal liver dullness, an X-ray film is often of great help by revealing an air bubble under both domes of the diaphragm. Leucocytosis, pericardial friction and signs of congestive failure appear later, and so are of no help at the onset of the emergency. Special consideration should be given to the following points:—

- (1) The fall of blood pressure is greater and more sudden in coronary thrombosis than in the shock of an acute abdominal catastrophe. If the patient's previous blood pressure is known, this sign may be very significant.
- (2) Arteriosclerosis of peripheral and retinal vessels is somewhat in favour of coronary thrombosis.
- (3) Dyspnoea, especially of Cheyne-Stokes type, suggests a cardiac cause.
- (4) The response to morphine. Though morphine will relieve the pain of intra-abdominal disease, it acts best when the cause is extra-abdominal.
- (5) The electrocardiogram. Although not invariably present in the early stages of acute myocardial infarction, the Pardee curve can be diagnostic even within an hour or two of the onset. The feature indicative of myocardial necrosis is displacement of the ST segment. The T wave originates directly from the downstroke of R in lead 1 ("early take-off") and the upstroke of S in lead 3 (anterior infarction). Sometimes this pattern is replaced by a dome-shaped ST segment with deep inversion of T especially in the chest leads. The

opposite pattern, depression of ST in lead 1, with elevation in lead 3, is produced by a posterior infarct. More pronounced deformities—deep, wide Q waves and elevated RS-T segments—may be seen in the direct precordial leads and are sometimes present when the standard leads appear normal. A normal curve even in the direct chest leads does not exclude a recent acute coronary occlusion. Abnormalities of the E.C.G. such as marked Q waves which are the permanent results of past trouble and unrelated to the present illness, must not be misinterpreted.

Dissecting aneurysm of the abdominal aorta.

This is rarely diagnosed accurately before death. The condition may be painless but more often it causes symptoms suggesting both perforation and myocardial infarction. Points which should arouse suspicion are that pain is overwhelming, widely distributed, and often radiates down both legs; the blood pressure remains high after the attack, and evidence of poor blood supply to the legs with weakness appears. When dissection occurs in the aortic arch the sudden fluctuating increase in superior mediastinal pressure may cause pulsation of one or other sternoclavicular joint. The pain is usually maximal at its onset and gradually improves in contra-distinction to the pain of coronary thrombosis which gets worse as time passes. Although shock may be profound, the abdominal signs are never quite like those of a major perforation. Liver dulness is not diminished, and X-rays show no gas bubble under the diaphragm. If there has been a previous attack an excessively calcified or "double aorta" may be seen on the radiograph.

Ruptured abdominal aneurysm or aorta.

After a period of retro-peritoneal leaking an aneurysm may suddenly burst into the peritoneal cavity and cause sudden severe abdominal pain and collapse. Early distension and rigidity may easily cause this catastrophe to be mistaken for perforation. Restlessness is a marked feature. If the doctor is forewarned by the knowledge that the patient had an aneurysm, he is fortunate. The condition is nearly always fatal in a short time and before arrangements for laparotomy can be made. Occasionally an aneurysm ruptures into the duodenum. Hæmatemesis is then a terminal event.

Abdominal angina.

This extra-pectoral form of angina presents as otherwise unexplained attacks of abdominal pain associated with a rise of

blood pressure in elderly arteriosclerotic patients. A tablet of glyceryl trinitrate 0.65 mg. (gr. $\frac{1}{100}$), under the tongue will often bring prompt relief. The more severe attacks probably result from minor infarctions of the bowel wall and eventually there may be a major mesenteric thrombosis.

Mesenteric vascular occlusion.

This condition may be heralded by mild "abdominal angina" but when the main superior mesenteric artery is suddenly obstructed by an embolus or thrombus, intense abdominal pain results. It is central and continuous; exacerbations are characteristic and they are often associated with vomiting. Signs of peristalsis soon disappear. Distension comes on next but there is no rigidity and little tenderness until later when peritonitis complicates the picture. Indeed, the contrast between the severity of the symptoms and the paucity of the abdominal physical signs is diagnostic. The patient is usually the subject of advanced cardiac disease (mitral stenosis, aortic valvular disease or coronary infarction) which might give rise to an embolus or there is arterial disease which might lead to thrombosis. Since gangrenous bowel always contains blood one would expect melæna—either spontaneous or after an enema—and this is sometimes a useful diagnostic point. Venous and arterial occlusion both lead to infarction but with a different march of symptoms. Arterial obstruction causes sudden symptoms and early shock whereas the symptoms of venous obstruction are less dramatic and run a slower course with shock only in the later stages.

Apart from minor cases, the prognosis is almost hopeless without operation and resection of the involved portion of the intestine, though a success following treatment with heparin has been reported. The condition is mentioned here because it has medical and surgical aspects and the cardiac condition should be recognised and treated.

Congestive cardiac failure.

This quite commonly presents with right upper abdominal pain and vomiting, leading us to suspect cholecystitis or a "quiet" perforation. Careful examination should reveal other evidence of cardiac failure. Sometimes there is a history of attacks of right hypochondriac pain following exertion, and presumably caused by liver distension.

Acute idiopathic pericarditis.

This uncommon condition may present as severe upper abdominal aching pain and the diagnosis may remain obscure until, after two or three days, a pericardial rub appears. It affects people of a younger age group than coronary thrombosis and so a cardiac cause of symptoms is apt to be forgotten and an E.C.G. which might suggest the diagnosis is omitted. Diagnosis in the early stages largely depends on awareness of the disease.

Pneumonia.

This is notorious as a cause of right iliac fossa pain resembling appendicitis (*see under Rigidity, page 65*).

Acute pancreatitis.

This condition is described here because, if diagnosis from the other emergencies can be confidently made, better results come from medical management (morphine and atropine, treatment of shock, etc.) than from operation. The patient is usually a middle-aged, obese subject, often with a history of gall stones. There is very severe epigastric pain radiating from left to right and sometimes to the back. Tenderness is very marked but there is not the abrupt onset or board-like rigidity of perforation. Shock is marked and sometimes fluctuating in degree. Vomiting is profuse and diarrhoea may occur. Slight icterus and a tinge of cyanosis (from involuntary inactivity of the diaphragm) are often present. Occasionally death takes place within an hour of the onset.

A plain X-ray film of the abdomen may demonstrate the useful fact that there is no gas under the diaphragm. The plasma diastase level is often markedly raised to levels 10 to 30 times the normal one of 70 to 200 Somogyi units per 100 ml. (The normal range for any particular laboratory should be known. The result can be given within an hour). High levels may occur in perforation (possibly from peritoneal absorption) and in intestinal obstruction. Morphine, renal failure and technical faults (contamination with saliva) may also cause raised diastase levels in the absence of pancreatitis but usually only 2 to 5 times the normal. If there is doubt, the test should be repeated but when one is confident that a surgical emergency is present, operation must be undertaken regardless of laboratory tests. Sometimes in a difficult case a small epigastric incision will reveal fat necrosis and so settle the diagnosis. Some would disregard any diagnosis of acute pancrea-

titis not proved by laparotomy or necropsy. A rare exception to this view would be the case in which subcutaneous fat necrosis appears later.

Pneumococcal peritonitis.

This is most often "seen in children and is dealt with on page 292.

Ascites.

In rare instances this is mysteriously fulminating and associated with diffuse abdominal pain. It is usually possible to demonstrate free fluid in the abdomen. Pyrexia and increased pulse rate are absent, so that observation rather than laparotomy and peritoneal aspiration to determine the nature of the fluid are justifiable. True peritonitis may be present as the concluding event in a series of tappings in hepatic cirrhosis.

Occasionally in a known case of tuberculous peritonitis acute symptoms may develop apart from those of an obstructive nature. They may be caused by a sudden increase in the number of tubercles and will settle down when treated with streptomycin and isoniazid.

The diabetic "acute abdomen."

Occasionally a diabetic passing into coma has severe epigastric pain. If the patient is not a known diabetic, the resemblance to a surgical acute abdomen may be very deceptive. Even when urine examination has revealed the presence of ketone bodies, an acute surgical condition cannot be excluded by this finding, since it may co-exist and, if it be inflammatory, it may have been the precipitating cause of coma. Nor will a white blood count be helpful since leucocytosis is usual in diabetic coma. A history of anorexia, nausea and drowsiness before the onset of pain points to a diagnosis of diabetes.

The need for differentiation is not very urgent. We have to decide between ketosis only and ketosis plus, for example, perforation. Operation should not be undertaken until ketosis is yielding to treatment. If the signs and symptoms are still present when ketosis has disappeared, some cause other than diabetes is present (*see also page 232*).

Pyelitis (*see also page 272*)

Acute pyelitis with pyrexia and pain in the right side of the abdomen may resemble appendicitis, but pyelitis usually causes

a higher temperature and greater leucocytosis. Examination of the urine reveals pus and organisms. Since these are almost normal findings in the "ordinary" specimen of urine in many women, care should be taken in assessing their significance and a catheter specimen obtained. Occasionally urinary abnormalities may be absent because of blockage of a ureter. In such cases, cystoscopy will help by showing an acutely inflamed ureteric orifice, and catheterisation will bring rapid relief of pain and pyrexia.

Great attention should be paid to the march of symptoms. Mid-line pain—vomiting—right-sided pain—fever—in this order are strongly suggestive of appendicitis. The absence of right-sided pain (of peritoneal origin) should not exclude appendicitis since the organ may be retro-cæcal or pelvic. Nor should dysuria necessarily be interpreted as a symptom of pyelitis since pelyic appendicitis may cause it.

It is said that the blood sedimentation rate is more likely to be raised in pyelitis than in unruptured appendicitis and this test may be helpful in a doubtful case.

Acute non-specific mesenteric lymphadenitis (Brennemann's syndrome).

This condition occurs especially in children of the bright, alert "rheumatic" type and causes recurrent attacks often mistaken for appendicitis and it is much more frequent. There is often a history of previous attacks. While differentiation may not always be sufficiently clear to justify expectant treatment certain points of difference should be noted. In lymphadenitis the face is flushed but not "toxic" as in appendicitis, the fauces are often inflamed but the tongue though red is hardly furred. The temperature is higher and vomiting earlier but rarer than in appendicitis. Diarrhoea practically never occurs. Pain is intermittent, severe and colicky and usually central. Tenderness also is more in the midline than in the right iliac fossa and sometimes two definite points of deep tenderness can be demonstrated. Shifting of the point of maximum tenderness, from altered direction of the pull on the mesentery when the patient lies on the left side for a time, suggests adenitis but is difficult to elicit. Psoas spasm, rebound tenderness, rigidity and rectal tenderness all of which may occur in appendicitis are absent in adenitis. Glands may be felt by careful palpation in the left lateral position with the knees flexed.

When symptoms persist, tuberculous adenitis should be suspected. A negative Mantoux test would exclude this and X-ray evidence of calcification would be in its favour. Glandular enlargement of known origin (*e.g.* glandular fever) is not usually associated with acute abdominal symptoms. If differentiation from appendicitis is not clear, the abdomen should be opened. Otherwise expectant treatment should be adopted. Occasionally the appendix has already been removed.

Acute hæmoperitoneum. (*see also* Mittelschmerz, page 115)

This may result when the amount of blood lost from a ruptured Graafian follicle is more than the usual minimal amount—so-called “apoplectic ovary.” There is sudden lower abdominal pain starting in one or other iliac fossa together with nausea but without pyrexia or a coated tongue. Tenderness is found on vaginal examination. An attack occurs typically midway between the menstrual periods. Symptoms disappear within a day or two. A similar picture may develop at any time if an ovarian blood cyst ruptures, a necrotic metastasis bleeds, or an aneurysm leaks. As the symptoms are more severe in these cases, surgical intervention is usually necessary.

General infections and toxæmias.

Abdominal symptoms may occur in the course of known infectious illnesses and suggest that some other condition is also present. We should be chary, however, of diagnosing an acute abdominal condition when there are general symptoms. It has been said that “an acute abdomen with a headache is never an acute abdomen.”

TONSILLITIS in children is often associated with abdominal pain and sometimes with true appendicitis. Appendicular pain and tenderness over the appendix may occur in the prodromal stage of SCARLATINA and MEASLES, but true appendicitis is not part of the picture of these diseases. A white blood cell count and a search for Koplik's spots are useful aids in diagnosis. PANCREATIC MUMPS causes pain and vomiting but no rigidity, and if parotid mumps is subsiding, diagnosis is easy. A history of contact is suggestive.

IN ACUTE RHEUMATISM severe, lancinating abdominal pain may occur. It is worse on movement and associated with tenderness. A history of acute rheumatism or chorea is usually obtainable. Polyarteritis nodosa may cause similar symptoms.

Vomiting is a common symptom of MALARIA and especially malignant tertian infections; in fulminating cases with visceral lesions there may be acute abdominal pain but there is no rigidity. The profuse sweating of malaria however (and also that of phthisis) may cause heat cramps (*see page 340*) of the abdominal wall so that an "acute abdomen" is simulated.

In INFLUENZA there may be griping abdominal pain in addition to headache and pharyngitis. Rigidity is absent and the extra-abdominal symptoms suggest the diagnosis.

ACUTE ALCOHOLISM may be complicated by abdominal symptoms but there is usually a good response to gastric lavage and intravenous fluids. Hourly observation shows progressive improvement.

Acute diseases of the spinal cord (*e.g.*, ACUTE POLIOMYELITIS) or nerve roots (*e.g.*, ZOSTER) may begin with abdominal pain. A period of watching will reveal typical signs (paralysis or a rash) of the true nature of the illness.

In rare cases when the small causal wound has been unrecognised, abdominal rigidity and pain have been the presenting signs of TETANUS (*page 481*). Toxins of industrial origin such as LEAD (*page 352* and CARBON TETRACHLORIDE (*page 351*) must be remembered as causes of acute abdominal pain. It must not be forgotten that lead may be ingested in many unusual ways and that proprietary preparations containing carbon tetrachloride for cleaning clothes may cause poisoning at home. The bite of certain SPIDERS (*page 472*) in the southern states of the U.S.A., has produced severe abdominal pain and rigidity.

Abdominal pain and vomiting are occasional manifestations of idiosyncrasy to OPIUM and MORPHINE, usually in large doses and especially in women. The mechanism is probably intense spasm of involuntary muscle in the biliary tract or elsewhere.

INFECTIVE HEPATITIS can cause pain and tenderness in the right upper abdomen with vomiting and moderate pyrexia resembling appendicitis. Careful study of the march of events and a search for bile in the urine and slight jaundice of the skin and conjunctivæ should point to the correct diagnosis.

TYPHOID may be confused with appendicitis, particularly in sporadic cases in children. The immediate diagnosis in an isolated case presenting with acute pain in the right iliac fossa

would be a matter for congratulation. Very few of these patients escape appendicectomy, but if a recent continental holiday or other feature brings the possibility of typhoid to mind the demonstration of a leucopenia may stay the surgeon's hand. A perforated typhoid ulcer may be very silent at first and present a little later as a medical emergency when gas under the diaphragm (as seen on X-ray examination) causes respiratory symptoms and spreading peritonitis creates the usual clinical picture.

Acute gastro-enteritis and ileo-colitis.

The diarrhoea and vomiting of gastro-enteritis may cause diffuse abdominal pain and tenderness. Care must be taken to exclude appendicitis, for it is too often forgotten that an inflamed appendix in the pelvis may itself cause diarrhoea. In a child the pain, tenderness and rigidity of appendicitis may be slight, and hence the need for great care in differentiating appendicitis from gastro-enteritis. Special attention should be paid to tenderness since it is a much more important feature of appendicitis than pain.

Ileo-colitis in children resembles intussusception because in both conditions there is colicky pain and in many cases, but not in all, bloody mucus may be passed. The essential features of intussusception are described on *page 291*; here only the differential points are mentioned. Passage of pure blood and clots does not occur in intussusception except in the rare case where an ulcerated Meckel's diverticulum forms the apex of the intussusception. Symptoms are less acute when the colon rather than the ileum forms the apex. Excoriation of the buttocks by evacuated succus entericus, and the peculiar smell which some can recognise, are in favour of ileo-colitis. Pyrexia may occur in both conditions but is more likely in ileo-colitis. Prolonged abdominal palpation, especially during a paroxysm, is important, and absence of the typical sausage-shaped tumour which contracts and relaxes is in favour of ileo-colitis. Microscopical examination of the faecal mucus shows numerous pus cells and a dysentery bacillus can often be grown on culture. The child with an intussusception is plump and healthy looking by contrast with the usual case of ileo-colitis, and it is remarkable how well he may appear between the paroxysms. Indeed, after the first sudden severe bout of pain, the subsequent attacks may be relatively mild.

Henoch's purpura (*see also page 179*)

In this condition colicky pain is caused by spasm of the bowel and infiltration of its wall with blood and serum, ("visceral hives"). Rigidity, vomiting, and distension are also present. Abdominal pain nearly always precedes the appearance of skin purpura, and until the latter appears the diagnosis may be in doubt. Joint pains and albuminuria are helpful differentiating points, and purpura induced in the arm by applying a tourniquet (Rumpel-Leede test) is suggestive. A past or family history of allergy should be sought. Even when skin lesions make the diagnosis certain it must not be forgotten that Henoch's purpura is not entirely a "medical" disease and that surgical complications such as necrosis and intussusception may arise.

Acute abdominal symptoms in blood diseases.

Acute abdominal pain has been described in polycythæmia (*page 175*); leukæmia [from perisplenitis (*page 175*)] and hæmophilia (*page 180*).

Allergy as a cause of acute abdominal symptoms.

Allergic abdominal symptoms are similar to those described under Henoch's purpura. Often there is a history that the attack began after eating some food such as pork or lobster to which the patient was known to be sensitive. If urticaria appears, the diagnosis is confirmed. The blood picture usually shows leucopenia with eosinophilia. Adrenaline by injection will alleviate the symptoms promptly. Cases have been reported of recurrent attacks of severe abdominal pain with pyrexia and signs suggesting appendicitis. Laparotomy in one such case revealed only great congestion of the subserous vessels.

Bornholm disease.

This condition (also known as Epidemic Myalgia) presents with mild pyrexia and pain usually in the chest and abdomen as the principal symptoms. It is thought to be due to a Coxsackie virus. Children are specially susceptible. An acute abdominal emergency may be simulated, but should be excluded by other features of Bornholm disease such as headache, myalgic pains elsewhere, tenderness of the costal margin, and leucopenia. Cough and vomiting—the usual accompaniments of acute thoracic and abdominal disease—are almost invariably absent and there is no

rash. When the disease is epidemic, the diagnosis is easy. Benign meningitis is an occasional complication.

Visceroptosis.

While true ptosis of abdominal viscera may be a cause of disability, the symptoms are usually of a chronic nature. Acute pain and vomiting are almost invariably attributable to the associated constitutional and nervous element. The patient complains of agonising pain, for which examination reveals no adequate explanation. Indeed, examination may be difficult on account of the commotion that the patient creates. Experience of these cases makes them easily recognisable. Morphine abolishes the symptoms completely but it is useful to give first 1 ml. of sterile water and record the effects of this placebo after 15 minutes; it may then be found that morphine is unnecessary. Nevertheless, such patients may develop appendicitis and other acute diseases, and if there is localised tenderness great care should be exercised and a leucocytosis sought.

Acute Porphyria.

Porphyrius are iron free pigments formed during hæmoglobin metabolism, but normally they do not appear in a free state. In certain hæmolytic diseases, in liver diseases, and after barbiturates have been taken, porphyrins may be found in the urine or they may appear without known cause.

We are only concerned here with acute porphyria. It is a rare condition four times commoner in women than in men. A family history of some manifestation of porphyria is often found. Acute attacks of severe abdominal colic occur, presumably from contraction of smooth muscle induced by the pigment. Many acute surgical conditions may be simulated, and the vomiting and absence of diarrhoea suggest intestinal obstruction. The symptoms are marked but are unsupported by signs and the belly is soft. There is usually a leucocytosis.

Associated findings—flaccid paralysis, mental changes and tachycardia (sometimes mistaken for hysteria)—should suggest the diagnosis. Confirmation depends on examination of the urine. This may be of normal colour when passed but if allowed to stand in the light, and especially if acidified, a colourless precursor, porphobilinogen, is converted into deep reddish-brown porphyrin pigments (the "window-sill" test). Tests for blood are negative and the abnormal pigment may be identified spectroscopically. Porphobilinogen is identified as follows: to 1 volume of urine add 1 volume of Ehrlich's aldehyde reagent and allow it to stand for 5 minutes, then add 2 or 3 volumes of a saturated solution of sodium citrate. Extract with 5 ml. of chloroform. If the pink colouration remains in the upper aqueous layer it is due to porphobilinogen, while if it is extracted into the lower chloroform layer urobilinogen is present. It is essential to neutralise all the HCl in the Ehrlich's reagent, as otherwise false positive reactions may be obtained as when indole acetic acid is present and is converted into pink-coloured uroscopin by acid. Treatment is purely symptomatic and the mortality is high. BAL (page 265) may cause

improvement. A.C.T.H. makes the symptoms worse. Some cases have responded to neostigmine (Prostigmin) 2.5 mg. subcutaneously every six hours.

Paroxysmal Myoglobinuria.

This is a rare cause of attacks of abdominal and generalised muscle pains which has led to mistaken laparotomy. The urine is very dark "like stout." It gives a positive guaiacum test for blood but red cells and porphobilinogen are absent. Spontaneous recovery is usual. (Berenbaum, Birch and Moreland. *Lancet*. 1955. 1. 892).

Abdominal pain at high altitude. (see page 392)

Abdominal Epilepsy.

There is some evidence that in epileptics and in patients suffering from focal lesions of the brain, short paroxysms of abdominal pain may occur from hypermotility of the bowel. Abdominal epilepsy, should not be thought of until the commoner causes of abdominal pain have been reviewed. If these are absent, abdominal pain in known epileptics or patients with cerebral tumours may be a manifestation of a focal brain disturbance, particularly if there have been previous attacks.

RIGIDITY

Abdominal rigidity of "medical" origin is usually caused by disease of the lungs and pleura, particularly in children (page 292). It is more a stiffness of the abdominal wall than the board-like rigidity of perforation, and some prefer to call it resistance. When vomiting is an early symptom, as in a child, the resemblance to true abdominal disease is great. The only medical condition causing real board-like rigidity is the spasm of tetanus but in this case tenderness is slight or absent.

Voluntary contraction of the abdominal muscles and abdominal rigidity of extra-abdominal origin can sometimes be made to relax by breath-holding and by pressure on the chest to restrict its movement. In this way any respiratory movement which the abdominal wall can make is brought out. The rigidity of extra-abdominal disease is a response to pain and, being central in origin, it diminishes after morphine. Rigidity in intra-abdominal disease persists after morphine.

Some unequivocal evidence of chest mischief such as a pleural rub or bronchial breathing often settles the matter. Slight cyanosis, inspiratory dilatation of the nostrils, raised respiratory rate, and the absence of rectal tenderness all point to the chest as the site of the trouble. Leucocytosis is higher in the initial stage of pneumonia than it is in appendicitis before perforation occurs.

Rigidity may be very marked in spontaneous pneumo-thorax and particularly hæmo-pneumo-thorax. Typical signs of pneumo-thorax (resonance with silence or distant amphoric breathing) or of air and fluid in the chest (splashing) are generally present. In a doubtful case, the chest should be X-rayed.

Diaphragmatic pleurisy.

This provides a special pitfall for the unwary for it causes referred pain in the abdomen, and resistance. If the central portion of the diaphragm be involved, pain may be referred to the shoulder tip. Physical signs are disappointingly few, but lack of movement of one side of the chest and dilatation of the nostrils on inspiration should point to the chest as the seat of the trouble. A rare but helpful symptom of pleurisy is persistent and painful hiccup. Other points in favour of pleurisy are a flushed face with a tinge of cyanosis, herpes of the lips, and a history of an initial rigor. Again, the march of events should be carefully scrutinised for the patient with diaphragmatic pleurisy or pneumonia is "too sick, too soon" to have appendicitis. Rarely, however, are his symptoms sudden enough to mimic a perforation. Sometimes an acute primary diaphragmitis produces a similar picture but without evidence of pulmonary disease.

Hæmatoma of the rectus muscle.

Debilitating diseases, senility, arterial disease, coughing, pregnancy, a lower abdominal scar and anti-coagulant therapy are all factors which predispose to bleeding into the rectus sheath. There may be premonitory soreness. Since the lower part of the rectus abdominis muscle has no posterior sheath, bleeding there may cause peritoneal irritation or may diffuse widely into the flanks. When a hæmatoma forms it can be shown to be in the abdominal wall because raising the head fixes the swelling. If the swelling increases in size, operation should be done to avoid the risk of massive hæmorrhage and infection.

VOMITING

When vomiting is the main symptom and pain is absent or atypical a "medical" cause is likely. Difficulty chiefly arises when vomiting is associated with pain and distension, also of medical origin. It must be emphasised that *pain with vomiting* is characteristic of small bowel obstruction, which should be

diagnosed before the distension appears. Hence, very great care must be taken before deciding that there is no obstruction.

Tabes dorsalis

The vomiting of gastric crises in tabes is a classical pitfall in diagnosis. Usually there is more retching than vomiting. Careful questioning will show that the epigastric pains have the same characteristics as those elsewhere, namely, they are lancinating and occur in rapidly recurring paroxysms. In an uncomplicated tabetic crisis, abdominal rigidity is absent. It is not sufficient to test the knee jerks alone, since a tabetic crisis can occur when knee jerks are normal if the dorsal rather than the lumbar posterior roots are mainly involved. The ankle jerks should be examined and analgesia to pin prick sought.

Acidosis.

Ketone bodies may be found in the urine of any child who has vomited frequently. They are the *result* and not the cause of vomiting whether this be "medical" or "surgical." The picture of cyclical vomiting is that of fever, vomiting and pale stools in a child who is highly strung and has had a diet containing excess fat. Often some infection such as tonsillitis starts off the attack. Rigidity is absent, and pain is probably caused by straining of the abdominal wall by the vomiting which precedes it. A careful study of the march of events will indicate that the cause of the ketosis is not surgical.

Acute poisoning.

The doctor should always keep at the back of his mind the possibility that the cause of the symptoms may be poisoning, or at any rate some ingested irritant. Patients nearly always try, however, to attribute their abdominal symptoms to some dietary indiscretion, and therefore we must be on our guard against accepting their explanation too readily and missing a true appendicitis. Poisoning by bacteria and their toxins, inorganic poisons, "green-apple colic" and acute alcoholic gastritis may all cause acute abdominal symptoms. "Bolus colic" may be suspected if a patient vomits a mass of obviously indigestible material.

Acholuric Jaundice.

Crises with abdominal pain and vomiting may occur in acholuric jaundice. The cause is biliary colic from "bile mud" produced by hæmolysis. Sometimes tenderness is discovered and suggests intra-peritoneal hæmorrhage. The other features of the case should make the diagnosis clear.

Uræmia.

Uræmia from chronic renal failure is a common pitfall for the surgeon. • The intense vomiting of sudden onset mimics high intestinal obstruction. The resemblance is greater if the patient is very thin, so that normal peristalsis is visible. The vomit is not fæculent as it may be in obstruction. Hæmatemesis from uræmic gastritis, or from bleeding gums may confuse the issue.

The general toxic appearance, the dry, furred tongue, and evidence incriminating the kidneys, such as a history of nephritis and hypertension, together with the presence of urinary abnormalities will indicate the correct diagnosis. In congenital cystic disease with uræmia, the kidneys are palpable. Peristaltic sounds are not increased in uræmic vomiting as they are in obstruction.

Sometimes the acute terminal pericarditis of uræmia causes upper abdominal pain, and the clinical picture closely resembles that of perforation.

Other medical causes of severe vomiting which may cause confusion are migraine (*page 219*), Ménière's syndrome (*page 224*), and the crisis of Addison's disease (*page 253*). Should the eye condition be overlooked the vomiting of glaucoma may be misleading.

DISTENSION

When this is of recent origin and is associated with vomiting it indicates intestinal obstruction which, incidentally, should have been diagnosed at the stage of yellow-brown vomit before distension appeared. The hernial rings must not be forgotten. Abdominal distension and pain may be a marked feature of acute hydronephrosis.

Occasionally great distension may complicate the picture of some other disease such as typhoid, pneumonia or ulcerative colitis, and make one wonder if a "surgical" cause is present. If the original diagnosis is firmly based, no difficulty should arise.

A rare condition, not very well known or understood, is that known as "bloating" or abdominal distension not due to gas. The distension is not present in the morning, but gradually appears as the day goes on, and may vanish suddenly without the passage of flatus. The intermittent nature of the swelling is emphasised in the picturesque name *Ventre en accordéon*. Lying on the back and flexing the thighs will cause the swelling to disappear, as will a dose of morphine. It is quite distinct from

pseudocyesis, and appears to be caused by lordosis plus descent of the diaphragm and cramp-like contraction of the abdominal muscles. Hence it is sometimes called hysterical abdominal proptosis. It occurs in psycho-neurotic women and occasionally in men.

Tight distension or rigidity plus distension is a late picture and indicates intestinal obstruction plus peritonitis. A rare cause of great abdominal distension in a young baby is an ano-rectal stricture. The distension is relieved by passing a catheter into the bowel, and cured by dilatation with the finger.

C. ALLAN BIRCH.

CHAPTER V

Other (Non-Surgical) Abdominal Emergencies

IN this chapter will be considered acute conditions affecting the abdominal organs which do not, as a rule, simulate acute surgical conditions, and which are not obstetrical or gynæcological.

VOMITING AS AN URGENT SYMPTOM

(For vomiting during anæsthesia see page 440)

Vomiting is a common and sometimes a presenting symptom in many urgent illnesses. The following classification is therefore given as an aid in reviewing possible causes in an obscure case. Obvious causes such as anæsthetics and pregnancy are not included. Usually some associated symptom or sign such as headache or papilloedema will point to the diagnosis.

CAUSES OF VOMITING.

1. Those acting on the vomiting centre.
Many acute infections (*e.g.*, Pneumonia) and acute specific fevers (*e.g.*, Scarlatina). Crisis of Addison's disease.
2. Those acting reflexly.
 - (a) Through the stomach itself.
Irritant poisons and unsuitable food.
Organic disease of the stomach.
In babies—congenital hypertrophic pyloric stenosis and habit vomiting.
 - (b) Through the abdominal and thoracic viscera.
Appendicitis. Peritonitis. Intestinal obstruction
Violent coughing from any cause.
 - (c) Through the nervous system.
 1. The brain. Increased intracranial tension from any cause. Meningitis. Migraine.
 2. The spinal cord. Gastric crises of tabes dorsalis.
 3. The special senses. Ménière's syndrome. Sea sickness. Unpleasant sights and smells.

Treatment.—This depends on the cause. Chlorpromazine (Largactil) 25 to 50 mg. intramuscularly three or four times a day is recommended. Iced champagne is reputed to "settle" the stomach. Gastric aspiration and lavage by the nasal route are temporary expedients in intractable cases (uræmia) or in those cases of unknown etiology.

ACUTE FOOD POISONING

General management.

Since this may be a communal as well as an individual emergency it is important to realise its implications at the onset and to act before evidence is lost. The following notes will help in deciding which food to suspect.

BACTERIAL FOOD POISONS.—Symptoms within two to four hours after eating the food, which is commonly cream cake or trifle containing staphylococcal toxin. On board ship it may be mistaken for seasickness.

BACTERIAL FOOD INFECTIONS.—Symptoms more than 12 hours after eating the food, which is commonly cream cake or trifle (dysentery organisms) or manipulated meats. Duck eggs unless boiled for 10 minutes may cause salmonella infections.

POISONOUS FOODS, e.g.:—Fungi taken in mistake for mushrooms; belladonna leaves accidentally included in dried herbs; narcissus bulbs eaten in mistake for onions and rhubarb leaves used as spinach. Foods sometimes poisonous include cheese, potatoes (especially if green and sprouting) and mussels which have fed on poisonous red plankton. The flesh of many warm water fish contains ichthyosarcotoxin, a neurotoxin which can cause severe symptoms.

EXOGENOUS CHEMICAL POISONS.—Salts of lead, zinc, arsenic and antimony may contaminate tinned foods or may be intentionally added to any food. Sodium fluoride, a kitchen insecticide, has been mistaken for baking powder.

- (1) Make a note of the foods eaten prior to the attack.
- (2) Take charge of suspected food—not forgetting left-over scraps and find out where it came from.
- (3) Save a specimen of vomit, urine and fæces for laboratory examination. Taking a rectal swab may save time if fæces are not available but fæces are more satisfactory.

- (4) Notify suspected food poisoning to the Medical Officer of Health and give him full facts of the cases.

Symptoms.—These are vomiting, diarrhoea and colicky abdominal pain. In shell-fish poisoning urticaria is common. Circulatory failure may be the main symptom in some cases.

Treatment.—If vomiting is troublesome, the stomach should be washed out with a gallon of warm water containing a tablespoonful of sodium bicarbonate. Copious bicarbonate drinks followed by vomiting induced by fingers in the throat may be used instead. After gastric lavage give castor oil, 1 fl. oz. for an adult, with orange juice if desired. A hot water bottle on the abdomen is comforting. Injection of Nikethamide 2 ml. may be needed.

When vomiting has ceased give fluids (fruit juice, diluted with half-strength physiological saline) by mouth. Some prefer an effervescent drink. In collapsed patients 5 per cent. dextrose in physiological saline may be given intravenously. Kaolin and Morphine Mixture N.F. 15 ml. ($\frac{1}{2}$ fl. oz.) may be given four hourly but morphine is best withheld until vomiting and diarrhoea abate.

Beware of giving a certificate of "Food poisoning" until proved. It may be wanted simply to blackmail a restaurant.

Tenesmus.

This means the urgent desire to empty the rectum, the act being accompanied by straining. It is a distressing symptom in some cases of diarrhoea. It may be caused by severe enteritis, impacted faeces, or even a foreign body. If rectal examination is negative, a starch mucilage enema with opium, as a palliative measure, may be tried. A heaped tablespoonful (15 G.) of starch is mixed with two tablespoonfuls (30 ml.) of cold water to form a smooth paste. To this is added, while stirring, one pint (568 ml.) of boiling water. After allowing to cool, 4 to 6 fl. oz. (120 to 180 ml., with 30 minims (1·7 ml.) of Tincture of Opium are given slowly as an enema, at body temperature.

Botulism.

This rare emergency is considered here because it is a form of food poisoning, although its symptoms are nervous and not abdominal. The causal organism, *Clostridium botulinum*, is present in soil, especially in California, but is rare in England. It and its toxin are destroyed by heat but the spores are unaffected. These

develop and produce toxin after a few days under anærobic conditions. Fresh food and recently made up dishes are therefore safe. Canned foods which have been boiled and those prepared by refrigeration are also safe. Both preserved meat and vegetables may be contaminated.

The characteristic symptoms which appear after a symptomless interval of 6 to 30 hours are diplopia, ptosis, fixed dilated pupils and dysphagia. They are due to the effects of toxin on the nervous system and may be gradual in onset. Pain and pyrexia are absent; the victim remains mentally alert. Slight initial nausea and vomiting, caused by proteolytic products, may occur.

Treatment consists in administering antitoxin intramuscularly and intrathecally but as this must be the correct type, laboratory help is essential in its use. The Ministry of Health, 23 Savile Row, London, W.1. (Tel., REGent 8411), or The Department of Health for Scotland, St. Andrew's House, Edinburgh (Tel., WAVErley 7241), should be consulted. Universal antidote (page 5) should be given, and followed by gastric lavage. Ether anæsthesia and alcoholic beverages are said to delay fixation of toxin in the tissues.

“MUSHROOM” POISONING

This is considered in detail because specific treatment is available. It is uncommon—only 39 fatal cases having occurred in England between 1920 and 1949—and so its very serious hazards are apt to be forgotten.

Many brilliantly coloured and peculiarly shaped fungi are not poisonous at all or are merely indigestible; they are generally avoided. The very poisonous Death Cap (*Amanita phalloides*), however, is easily mistaken for the edible mushroom.

Tests of edibility.

Most of the popular tests are fallacious. The edible mushroom peels easily, but so does the Death Cap. Some fungi change colour alarmingly when cut, but this does not indicate that they are poisonous. Failure to blacken a silver spoon during cooking is no proof of edibility. The fact that fungi are eaten by rabbits does not mean that they are safe for man, as rabbits can eat the Death Cap with impunity. Hence it is only by recognising poisonous fungi on sight that emergencies can be avoided.

The Death Cap (*Amanita phalloides*). (Fig. 7)

The cap of this fungus is smooth and yellowish olive, streaked with darker fibrils, but varies in colour. The gills, however, are permanently white and this is an important distinguishing feature as the gills of the edible mushroom are never white



FIG. 7

The Death Cap (*Amanita phalloides*). Gills white; volva at base of stem; cap yellowish-green. (Height, $4\frac{1}{2}$ in.; width of cap, $3\frac{1}{2}$ in.; width of stem, $\frac{1}{2}$ in.)

(Fig. 8). The base of the stem of the Death Cap is surrounded by a persistent cup or volva but this may be missing if the whole fungus is not gathered. No volva is present on the stem of the edible mushroom.

SYMPTOMS.—From the point of view of treatment, cases of "mushroom" poisoning fall into two groups:—(1) Those with early symptoms. (2) Those with delayed symptoms.

Early symptoms.—These are diarrhoea and vomiting, and may be caused by many fungi and even edible ones if not fresh. or if eaten by allergic subjects. Sometimes the early symptoms are nervous—delirium and hallucinations—as from eating *Amanita muscaria*, a fungus whose cap is red with white patches.

Delayed symptoms.—These come on after a latent period of eight hours or more and this delay is characteristic of Death Cap poisoning. After this interval, vomiting and diarrhoea with intense abdominal pain begin. These symptoms may abate a little, but collapse and death occur in at least half the cases from liver cell function failure.



FIG. 8

Edible mushroom (*Psalliota campestris*). Gills brownish-purple ; base of stem clubbed ; no volva. (Height, $3\frac{1}{4}$ in.; width of cap, 5 in.; width of stem, 1 in.)

Treatment.—The decision whether to treat for Death Cap poisoning or not may be made by:—

- (1) Examining the uneaten fungi.
- (2) Considering the time interval between ingestion and symptoms.
- (3) Examining the vomit and faeces for spores. Although Death Cap spores could not be distinguished from all other spores they are sufficiently characteristic to confirm a diagnosis. They are colourless subglobose bodies measuring 8 to 11 by 7 to 9 μ and contain a large oil drop (Figs. 9 and 10).

If in doubt treat for the more serious conditions as follows:—

- (1) Give atropine 1 mg. (gr. $\frac{1}{60}$) intravenously to an adult. This is the antidote to muscarine-like alkaloids. Injection of Nikethamide 2 ml. repeatedly may be needed for collapse.
- (2) Wash out the stomach (page 537) and leave in it 60 ml. (2 fl. oz.) of White Mixture of Magnesium Sulphate B.P.C. This treatment will probably be too late in Death Cap poisoning but it would be wise to consider its use in every case.

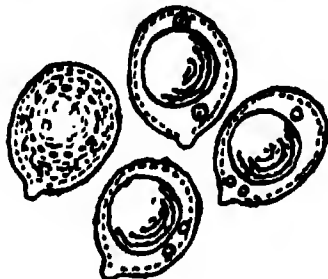


FIG. 9

Spores of the Death
Cap (*Amanita phal-*
loides). $\times 1,000$.

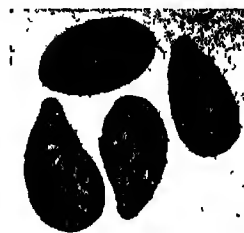


FIG. 10

Spores of the Edible
Mushroom (*Psal-*
liota campestris).
 $\times 1,000$.

- (3) Empty the bowel by enema.
- (4) Start energetic treatment as described under Hepatic Failure (page 82) including cortisone or A.C.T.H.
- (5) Obtain and administer antiphallenic serum (page 620).

The dose will be indicated on the amount sent. It is usually about 40 ml. and should be given intramuscularly or intravenously with the usual precautions (page 579).

- (6) Give the rabbit stomach-brain treatment of Limousin and Petit. This is based on the fact that while cats die after eating *Amanita phalloides*, rabbits do not. But the juice of *Amanita phalloides* injected subcutaneously into rabbits is fatal, suggesting that the toxin is destroyed or neutralised in the rabbit's stomach. Cats fed on *Amanita phalloides* plus rabbit's stomach, survive for several days. If rabbit's brain is given also, they recover completely.

The treatment recommended is to give up to five uncooked rabbits' brains and stomachs minced up, daily for several days. Its practicability in a vomiting patient seems questionable, but the emergency is so desperate that it should be tried. All those who have shared the meal containing fungi, irrespective of

whether they have symptoms or not, should submit to stomach lavage.

Bowel lavage is of doubtful value since fungi are unlikely to have reached the colon. A more effective measure, after



FIG. 11.

Deadly Nightshade (*Atropa belladonna*). Yellow variety on right.

gastric lavage and leaving White Mixture of Magnesium Sulphate B.P.C. in the stomach, is to repeat ounce doses of Epsom salts and drinks of hot tea every half hour until the bowels act.

BELLADONNA POISONING

Every year at blackberry time, the country doctor is liable to meet belladonna poisoning. The patient is usually a child out for a day in the country. The berries eaten are those of the Deadly Nightshade (*Atropa belladonna*) (Fig. 11), like small black cherries, but those of Woody Nightshade or Bittersweet (*Solanum*

dulcamara) like red currants, or of Black Nightshade (*Solanum nigrum*) like black currants, may be responsible.

The Thornapple (*Datura stramonium*) (Figs. 12 and 13) when ripe bursts open to reveal numerous small black seeds which are attractive to children. They contain $\frac{1}{2}$ per cent. of hyoscine and



FIG. 12

The Thornapple (*Datura stramonium*). Scale in inches.

hyoscyamine and so cause symptoms similar to those of belladonna poisoning.

Some hours after eating these berries or seeds, the child is found confused and excited and has a flushed face and dry mouth. He is "hot as a hare, blind as a bat, dry as a bone, red as a beet and mad as a hen." The widely dilated inactive pupils give him

a startled appearance. The heart is rapid and the bladder distended, presumably from spasm of the external sphincter.

It is best to use an emetic of salt (2 tablespoonfuls to a tumblerful of water) or mustard (1 tablespoonful to a tumblerful of water) rather than a stomach tube as the berries may block this. Apomorphine is not a very effective emetic in belladonna poisoning.

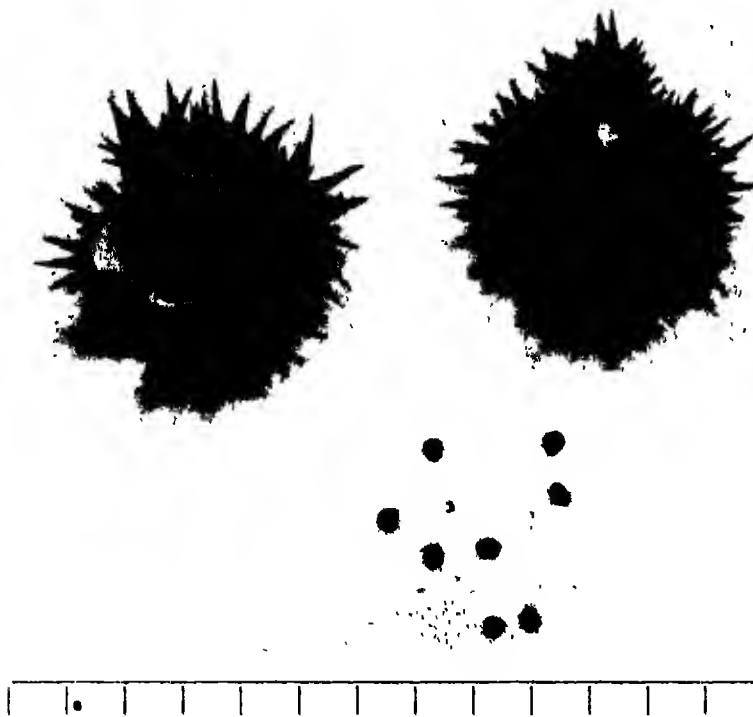


FIG. 13

Fruit and seeds of the thornapple (*Datura stramonium*). Scale in inches.

The specific antidote is neostigmine (Prostigmin) 0.5 to 1.0 mg. intramuscularly. The excitement should not be treated with morphine since this might enhance the depression which will follow in any case. Sodium phenobarbitone 65 to 200 mg. (1 to 3 grains) by intramuscular injection is preferable. Similar symptoms may result if belladonna enters the body in excess from liniments, plasters, medicines and eye drops.

In a doubtful case a delicate test is to instil a few drops of the child's urine into a cat's eye. If belladonna is present rapid dilatation results.

POISONOUS PLANTS

Anxious parents may telephone the doctor urgently for advice when their child has eaten some odd berries. Knowledge of the

plant concerned can often enable a reassuring message to be given, but the patient should be seen.

Mistletoe berries cause no trouble unless large numbers are eaten. Holly berries, at the worst, cause purgation. The fruits of honeysuckle, wistaria, ivy and laburnum can cause alarming but not fatal symptoms. Some members of the parsley family, *e.g.*, common hemlock, contain coniine and are very poisonous. Yew leaves and seeds contain large quantities of a poisonous alkaloid taxine but the pulp of the berry is innocuous. Gastro-intestinal symptoms may result if a yew leaf is accidentally cooked with vegetables. Monkshood (*Aconitum napellus*) whose roots may be mistaken for horseradish is, perhaps, our most poisonous plant. Altogether some 200 British plants contain poisonous constituents; poisoning from eating them will usually have to be treated on general lines.

BILIARY COLIC

Biliary colic most commonly results from distension of the biliary tract, due to spasm at the ampulla of Vater, caused by the impaction there of a gallstone. It is one of the most severe pains known. It is a visceral pain and so is felt centrally at first but soon radiates to the right hypochondrium and right scapular region. It comes and goes at intervals of several minutes and with each paroxysm the patient rolls about and is doubled up in agony and sweats profusely. Vomiting may occur at the end of an attack, which may last from a few minutes to several hours and leave the patient limp, pale and sweating. Similar but less severe pain may be due to widespread spasm in the biliary passages and occurs as a sequel to old-standing cholecystitis and chronic cholangitis, irrespective of whether the gall-bladder has been removed or not.

Examination between the paroxysms reveals a tender liver and resistance of the upper right rectus muscle but little else. Marked local tenderness and rigidity suggest peritoneal pain from cholecystitis. Evidence of slight jaundice—clinical or bio-chemical—helps to clinch the diagnosis. When colic complicates acholuric jaundice pigmentation is obvious. When gall-stones have formed, as they may do in long-standing cases of acholuric jaundice, colic may be associated with true obstructive jaundice should a stone block the common bile duct. Bile will then appear in the urine.

Differential diagnosis.

Coronary thrombosis may be simulated (*see pages 53 and 165*). Acute pancreatitis (*page 57*) may be an associated condition. Other abdominal colics are usually less severe and have their own characteristic distribution and accompaniments.

Treatment.—Which of the several available remedies is used will depend on the circumstances. Mild attacks will respond to most analgesics.

- (1) Pethidine 100 mg. intravenously.
- (2) Morphine 15 to 30 mg. (gr. $\frac{1}{4}$ to $\frac{1}{2}$) intravenously. Morphine relieves pain by a central action and may aggravate the pain by causing spasm of the sphincter of Oddi. It is well to combine it with hyoscine gr. $\frac{1}{100}$. Large doses cause respiratory depression but this can now be avoided without lessening the analgesic effect by giving 15 mg. of Amiphenazole (Daptazole) after the dose of morphine.
- (3) Glyceryl trinitrate 0.65 mg. (gr. $\frac{1}{100}$) under the tongue. (Amyl nitrite should be avoided because of the unpleasant flushing and palpitation it causes).
- (4) Aminophylline (*see page 598*).

Very occasionally anæsthesia induced by intravenous thiopentone is necessary. Heat locally and a strong carminative mixture may help.

HEPATIC FAILURE ("CHOLÆMIA")

Liver-cell failure is an uncommon but serious emergency. It most commonly occurs in virus hepatitis of which it is a very rare complication, and in portal cirrhosis when precipitated by gastrointestinal bleeding, operations and alcohol. It can complicate many other liver disorders such as amœbic and leptospiral hepatitis and the liver damage occurring in thyrotoxicosis, septicæmia and poisoning by fungi, drugs, anæsthetics and industrial solvents. The sudden (surgical) release of biliary tract obstruction may precipitate it.

Because of the many causes the symptoms will vary but as the damage is often reversible the warning nervous signs and foetor hepaticus common to all of them should be recognised. At first there are minor alterations in behaviour; a previously cheerful, well-behaved patient becomes awkward and truculent. The nurse should be warned to report such changes. Confusion, mania and

deep coma may follow. There is a characteristic "flapping" tremor of the outstretched hands which may be easily missed unless specially looked for. Foetor hepaticus, like the smell of a freshly-opened corpse, is a characteristic, though not necessarily ominous sign. All these signs are probably caused by absorption from the intestine of substances which a normal liver can detoxicate. (They may all occur also, particularly after protein feeding, when blood from the intestine by-passes the liver through a porto-caval anastomosis).

Renal damage, as shown by oliguria, albuminuria and hæmaturia may complicate the picture and results, not only from disease known to affect both organs but also, as a terminal event from toxins arising in the liver ("hepato-renal syndrome"). When hepatic failure complicates operation on the biliary tract there is usually high fever, jaundice, anuria and coma. It probably results from a fulminating infection in an already damaged liver ("hyperpyrexia death").

Treatment.—This has two aims (1) to protect the liver cells and (2) to eliminate toxins arising in the intestine.

Protection of liver cells.

Not all the remedies mentioned have the fullest scientific basis but as they are without risk and offer hope of saving the patient their use should be considered.

- (1) **GLUCOSE.** The liver is protected from the action of toxins when it contains adequate stores of glycogen provided by glucose. At least 500 G. should be given in 24 hours either by stomach tube or as 25 per cent. dextrose intravenously. To prevent thrombophlebitis in peripheral veins strong dextrose solutions should be given into the innominate or other large vein by polythene tube. Too rapid infusion will cause much to be lost in the urine.
- (2) **POTASSIUM.** As large amounts of dextrose intravenously can cause severe hypopotassæmia 0.5 G. potassium chloride should be added to each 500 ml. of 25 per cent. dextrose.
- (3) **INSULIN.** This aids deposition of glycogen in the liver and muscles and should be given in doses of 10 units for every 50 G. glucose retained.
- (4) **CALCIUM.** Inject *very slowly* into the vein or infusion tube 10 ml. of 10 per cent. calcium gluconate every 4 hours or give it intramuscularly.

- (5) **VITAMINS.** Give intravenously each day 200 mg. aneurine (4 ml. Benerva, Roche); 300 mg. Nicotinamide (6 ml. Nicotinamide, Roche) and 150 mg. riboflavin (30 ml. Bflavit, Roche). Parentrovite (Vitamins Ltd.) is a proprietary preparation of these and other vitamins. A pair of 5 ml. ampoules provides similar dosage. Vitamin K (Konaktion, Roche) 10 mg. should be given by mouth or intravenously each day.
- (6) **α -TOCOPHEROL** (Ephynal, Roche). Give 300 mg. each day, intramuscularly at first but by mouth as soon as possible as it is more effective by this route.
- (7) **BLOOD TRANSFUSION.** The objects of this are to increase blood proteins and to correct anæmia. If epistaxis, melæna, or other hæmorrhage occurs *fresh* blood should be transfused.
- (8) **GAMMA GLOBULIN.** Although of most value when given within seven days of exposure to a case of infective hepatitis it may mitigate hepatic failure due to this cause. Up to 10 ml. should be injected intramuscularly (*see page 515*).
- (9) **SODIUM GLUTAMATE.** The level of ammonia in the blood is known to be raised when liver function is impaired and it or some derivative may be a factor causing coma. Because glutamic acid normally combines with intracellular ammonia in the brain its use has been suggested in treatment. Results have been inconsistent. Up to 50 G. of sodium glutamate B.D.H. can be used in 24 hours. A 25 per cent. solution should be added to 500 ml. of 5 per cent. dextrose and given intravenously very slowly. This dose should not be exceeded in 24 hours. Glutamic acid 20 G. daily (in 0.5 G. tablets) should be given by mouth afterwards.
- (10) **Cortisone and A.C.T.H.** Cortisone (in intramuscular doses of up to 1,000 mg. daily) or A.C.T.H. (in intravenous doses of 25 units several times a day) are thought to have caused recovery in a few cases of hepatic coma.

Elimination of intestinal toxins.

As nitrogenous compounds give rise to toxins causing neurological symptoms in these cases protein feeding should be avoided until the patient improves. Even then the protein intake should not exceed 50 G. daily. The calorie needs must be supplied as glucose. As bacterial activity in the bowel may increase the release

of toxic products tetracycline 1.0 G. daily should be given.* Daily bowel action should be ensured by the use of a laxative.

An intact liver is necessary for the detoxication of morphine and barbiturates and so these drugs should be avoided in hepatic failure. Paraldehyde is preferable (*see page 597*).

RECTAL PAIN (PROCTALGIA FUGAX)

An urgent call for the doctor may be occasioned by an attack of boring unremitting cramp-like pain in the rectum just above the anal sphincter. It begins as a slight pain and works up to a maximum in five or ten minutes but does not radiate. Sometimes it is associated with sexual intercourse. There is no diarrhoea or passage of flatus. A fainting attack may complicate the picture. Local examination is usually negative but sometimes reveals a tender band possibly the result of spasm of the levator ani. It should always be made since similar pain may result from impacted faeces.

The taking of food and drink often brings prompt relief presumably because the gastro-colic reflex inhibits the painful spasm. Other remedies are a small enema, a finger in the rectum and pressure on the perineum (sitting astride the cold edge of the bath). Of the many drugs that have been used pethidine 100 mg. by mouth is probably the best.

PRIAPISM

This rare condition is a pathological persistent erection of the penis in the absence of libido. Erection persists because of thrombosis in the corpora cavernosa or deep dorsal vein of the penis. Recurrent incomplete priapism is usually associated with diseases of the nervous system, *e.g.*, disseminated sclerosis and then retention is a common complication. Supra-pubic drainage is better than a catheter which may cause gangrene. In purely vascular priapism the glans and corpus spongiosum are normal and so retention does not occur. Leukæmia should be suspected. Sedatives should be given and anti-coagulant therapy (*page 595*) started particularly in a case due to trauma. Attempts to obtain relief by intercourse will fail.

ACUTE LUMBAGO

By acute lumbago we mean a sudden severe low back pain which immobilises the patient. His back becomes "locked." When this follows immediately on a sudden strain such as lifting

a heavy object in the flexed position, a surgical rather than a medical emergency may be suspected. The pain may, however, appear more gradually after a period of stooping as in gardening and constitute an extremely inconvenient situation if not a dire emergency (*see also* Drowning, page 120).

Treatment.—The patient should be put to bed (non-sagging)—often a long and difficult process—and given an analgesic such as three Compound Tablets of Codeine B.P. or their equivalent. Warmth should be applied in whatever form is most convenient. An electrically heated pad to lie on is very comforting. If pain is very localised it may be relieved by infiltration of the affected area with 1 per cent. procaine containing 0·005 per cent. adrenaline hydrochloride.

Recurrent attacks, or the presence of even slight pain in the distribution of the sciatic nerve make it probable that the “locked” back is due to a protrusion of an intervertebral disc. In such a case the question of more effective early immobilisation than bed rest, such as by a corset or plaster jacket, should be considered.

C. ALLAN BIRCH.

HÆMATEMESIS AND MELÆNA

The introduction of liberal feeding and drip blood transfusions have greatly improved the prognosis of bleeding from a peptic ulcer in recent years. The licking of dry parched lips and the deathly pallor of extreme anæmia are seldom seen in hospital wards to-day. Nevertheless, this emergency is still serious, particularly in patients over 50 years and when associated with other complications.

Etiology.

Peptic ulcer is the most common cause of gastro-duodenal hæmorrhage. Bleeding may occur from a small superficial acute ulcer or from a large chronic crater eroding the pancreas, and from all gradations between these extremes. All may cause severe and repeated hæmorrhage, but while the mortality from an acute ulcer is low (2 per cent.), that from a chronic ulcer is as high as 20 per cent. Many acute or subacute ulcers heal quickly and may come and go within a few weeks. Consequently nearly a third of all admissions do not show any radiological evidence of ulcer when examined two to four weeks later.

Nevertheless, their presence has been shown in many such cases by early gastroscopy. Of 673 consecutive cases of hæmatemesis and melæna 615 were found to be due to peptic ulcer.

Both carcinoma of the stomach and cirrhosis of the liver are relatively uncommon causes of gastro-duodenal hæmorrhage. Bleeding may sometimes be due to traumatic laceration of the cardio-oesophageal junction from the strain of severe vomiting.

Treatment may be discussed under four headings—(1) feeding, (2) blood transfusion, (3) surgery, and (4) oesophageal tamponade.

Feeding.

There has been much controversy in recent years on the advisability of liberal feeding. With the traditional starvation treatment there was a mortality rate of about 15 to 25 per cent. in all series of a hundred or more admissions. Meulengracht reported, in 1934, a 2 per cent. mortality in Copenhagen after liberal feeding. Admittedly, previous Danish figures showed only a 7 per cent. mortality (perhaps there are more acute peptic ulcers in Denmark). Adoption of his recommendations on diet in this country has been accompanied by a fall in mortality which is now between 5 and 10 per cent. The improvement has also coincided with the use of drip blood transfusion which has certainly contributed, but probably the better figures are due mainly to the elimination of the deaths from dehydration. Nearly half the patients treated by starvation and restricted fluids died without blood being present in the gut. They died of renal failure from dehydration 7 to 10 days after admission. It is not necessary to give liquids only and a nutritious semi-solid diet will provide adequate fluid. Many clinicians favour a puree diet for it replenishes the depleted body proteins and re-stocks the liver with glycogen. The patient is, therefore, in a stronger position should bleeding recur. There is no evidence that early feeding increases the risk of further hæmorrhage and clinically the patients do well.

Blood transfusion.

The introduction of blood banks and the Medical Research Council standardisation of "giving sets" has greatly simplified the administration of blood. Nevertheless, unpleasant reactions may still follow the most careful transfusion. It is therefore necessary to appreciate the exact purpose for which it is given. It is

very uncommon for a patient to bleed to death from the initial erosion of the blood vessel. In spite of a most profuse loss of blood and severe shock, recovery is known to take place without transfusion. There is a risk that in some older patients prolonged shock may cause irreversible cellular changes so that death supervenes a few days later. But death is more often the result of the second, third, fourth or fifth hæmorrhage within the course of as many days. Blood transfusion after the initial hæmorrhage provides a reserve should bleeding recur—i.e., it leaves him with a greater margin of blood to lose before the anoxæmic level is reached, and establishes a more favourable position should bleeding start again. There is no evidence that *slow* blood transfusion increases the likelihood of further bleeding. If women have to be transfused during the reproductive period of life they *must* be tested for Rh. agglutinogens and receive Rh. negative blood if they are Rh. negative. Men requiring repeated transfusions should be dealt with similarly.

Surgery.

The patients who are likely to die under medical treatment are those with known chronic ulcers whose bleeding recurs during the first few days after admission to hospital. The mortality from medical treatment is about 50 per cent. for those over 50 who have such *repeated* bleeding from a chronic gastric ulcer. Persistent ulcer pain after admission and thickened tortuous arteries are bad prognostic points. It is in this group that operation (partial gastrectomy) should be considered. A laparotomy may be needed also for continued bleeding in the absence of previous ulcer symptoms, in order to exclude a simple tumour of the stomach, a painless chronic peptic ulcer or a Meckel's diverticulum. A small barium meal or gastroscopy may be undertaken in this group before laparotomy.

Occasionally a patient may be found to have pyloric stenosis with visible peristalsis. If this is the case it is advisable to empty the stomach at night and arrange operation as soon as the general condition allows.

Bleeding from portal hypertension.

The past history will often indicate the possibility of portal hypertension and the presence of an enlarged spleen, or finding of spider nævi in the upper part of the body may confirm the diag-

nosis. Bleeding does not necessarily come from rupture of the œsophageal varices but may arise from acute gastric ulcers eroding engorged gastric veins. Raising the foot of the bed may increase the rate of blood flow in the œsophageal veins and thereby diminish the lateral pressure on their walls and lessen the hæmorrhage. This simple measure is always well worth trying.

Another simple method is to try to promote clotting with thrombin (Thromboral, Maw). As its action is impeded at pH below 6·5, 50 ml. of M/7 phosphate buffer* should be given first and then about 5 minutes later 20,000 units (2 G.) of Thromboral dissolved in 30 ml. of buffer should be sipped slowly. This dose should be repeated every 2 or 3 hours. Failing the buffer, milk could be used. Mechanical pressure on the veins by a balloon in the œsophagus is worth trying in severe cases. (For technique, *see page 614*).

An emergency porto-caval anastomosis for bleeding from portal hypertension has been successfully performed and in suitable patients in whom the liver function is known to be good, this should be kept in mind.

MANAGEMENT OF GASTRO-DUODENAL HÆMORRHAGE

1. Reassure the patient.
2. Secure mental relaxation with morphine 11 mg. (gr. $\frac{1}{6}$) or injection of sodium phenobarbitone 0·2 G. (gr. 3).
3. Unless there is shock, allow one to two pillows as desired.
4. If there is shock, raise foot of bed and give oxygen at 6 litres a minute by a mask (*see page 573*).
5. Order hourly pulse chart.
6. Take about 14 ml. of blood. Put up to 5 ml. in an oxalate bottle for blood urea and hæmoglobin estimation; up to 5 ml. in a plain bottle for serum for grouping and use the rest to make up the special oxalate tube for prothrombin activity to 2·5 ml. exactly.

Note:—

- a. The hæmoglobin may remain high for 6 to 18 hours after bleeding and so may be misleading.

20·4 G. anhydrous disodium phosphate (Na_2HPO_4) in 1,000 ml. water and 1·95 G. anhydrous dihydrogen potassium ortho-phosphate (KH_2PO_4) in 100 ml. water produce 1,100 ml. of seventh molar phosphate buffer of pH 7·6.

- b. A blood urea of 70 to 100 mg. per cent. is common after a brisk hæmorrhage. If over 150 mg. per cent., consider the possibility of dehydration (*see page 463*), chronic nephritis or alkalosis (*see page 274*) and give fluids intravenously.
7. Arrange for blood grouping of available relatives if there is no blood bank.
 8. Allow dilute saline (one part physiological saline to two parts water) by mouth as desired in a feeding cup by the bed.
 9. Order semi-solid diet, *e.g.*:—
 - 6 a.m. Cup of milky tea.
 - 8 a.m. Porridge and Bemax or lightly-boiled egg; thin bread and butter and jelly marmalade; cup of milky tea.
 - 10 a.m. Cup of milk and biscuit.
 - 12 noon. Minced meat, chicken or steamed fish; mashed potato; puree carrot or cauliflower.
 - 2 p.m. Egg custard or cereal pudding or apple puree; orange juice.
 - 4 p.m. Cup of milky tea; three slices of thin bread and butter; bramble jelly; sponge cake.
 - 6 p.m. Cream of vegetable soup or minced chicken sandwich.
 - 8 p.m. Milk pudding or cup of milk.
 - 10 p.m. Cup of milk and biscuit.

Give milk feeds during the night if awake.

If the patient cannot tolerate food (this is rarely so), or if there is pain or clinical evidence of pyloric stenosis. give 7 fl oz. milk feeds two-hourly.
 10. If there is clinical evidence of a massive hæmorrhage or if the hæmoglobin is below 50 per cent., give a blood transfusion at 40 drops a minute. It is rarely necessary to cut down on a vein.
- Note:*—The appearance of the patient immediately after a brisk hæmorrhage may be very misleading. The lips and face may be very pale from reactive vaso-constriction. The colour, and the initial fall of blood pressure may quickly improve. A review after 20 to 30 minutes may help in assessing the severity of the hæmorrhage. It is generally

unwise to give the blood quickly (*see page 44*) but rapid transfusion may be necessary after massive hæmorrhage. If the patient is already profoundly anæmic it is important to transfuse slowly and to watch the jugular venous pressure. Packed red cells are preferable for transfusions when the hæmoglobin is below 35 per cent. The packed cell volume (P.C.V.) (hæmatocrit reading or volume of packed cells per 100 ml. of blood—normally 47 per cent. in men and 42 per cent. in women) is a guide as to how much blood is needed, if estimated after post-hæmorrhagic dilution has occurred. Roughly speaking, the packed cells of 500 ml. of blood will raise the P.C.V. by 5 per cent. and the Hb by 8 to 10 per cent.

11. Antacids may be given. Aluminium hydroxide emulsion $\frac{1}{2}$ fl. oz. (15 ml.) two-hourly is recommended as this has an anti-peptic as well as an acid neutralising action and so may hinder the digestion of clot. Double doses at night may be given while awake. After three days it is sufficient to give $\frac{1}{2}$ fl. oz. (15 ml.) four times a day or magnesium trisilicate, or tribasic alkaline powder (equal parts of calcium and tribasic magnesium phosphate).
12. Give ascorbic acid, 200 mg. three times a day for five days and then 50 mg. daily (or orange juice). Ascorbic acid deficiency retards healing of the ulcer.
13. Ensure sleep at night with chloral hydrate 2 G. (gr. 30) well diluted, or sodium amytal 0.2 G. (gr. 3) or soluble barbitone (Medinal) 0.5 G. (gr. $7\frac{1}{2}$).
14. The bowels are usually inactive for several days after a hæmorrhage. It is unwise to give aperients or early enemas. Reassure the patient and give a simple enema on the fourth day.
15. Give compound ferrous sulphate tablets B.P.C. 0.2 G. (gr. 3), three times a day, preferably crushing the tablet before swallowing.
16. Arrange bed exercises for the patient. Allow him to get up for a short time as soon as possible but not to leave hospital until the hæmoglobin is 60 per cent. and the stools negative for occult blood.

SPECIAL INSTRUCTIONS CONCERNING THE SERIOUSLY ILL PATIENT

The milk drip.

For continued bleeding when surgery is not indicated, *e.g.* in a young person with a known or a probable duodenal ulcer, it is worth while using a milk drip. Pass a lubricated Ryle's tube through the nose as far as the second mark. Join by a connection chamber to rubber tubing and a douche can or an inverted transfusion bottle. The rate of flow can be adjusted by a screw clip on the tubing. For preference 5 to 6 pints should be given in 24 hours (the average drip bulb *kept* at one drop every second will deliver about 8 pints in 24 hours), but if the milk supply is inadequate give two pints during the night and revert to light diet during the day.

Gastric lavage.

However severe a hæmorrhage may seem, and however unpromising the prognosis, always persist with treatment. Occasionally emptying the stomach and washing out with water may tip the scales in the patient's favour.

Oxygen.

An oxygen tent or oxygen at 6 litres a minute through a mask may greatly assist a desperately ill patient.

Saline.

If there is no available blood, give intravenous dextran or 5 per cent. dextrose in physiological saline. Plasma is best avoided because of the risk of jaundice; saline is probably just as beneficial in acute hæmorrhage. Should the general condition or high blood urea call for increased intake of fluid, non-saline infusions should be given intravenously to avoid retention of salt with resulting hydræmia and oedema. 5 per cent. dextrose made up with sterilised water for intravenous use should be given alternately with 5 per cent. dextrose in physiological saline for the first day, and subsequently the saline reduced to every third bottle.

Vitamin B deficiency.

Acute vitamin B deficiency causing heart failure or coma from petechial hæmorrhages in the brain may be precipitated by giving dextrose intravenously to a malnourished patient.

This may be prevented by giving aneurin 50 mg. (1 ml. B₁₂nerve, Roche), nicotinamide 200 mg. (4 ml. Nicotinamide, Roche), and riboflavin 10 mg. (2 ml. B₂flavit, Roche) daily into the dextrose drip.

Vitamin K deficiency.

If this is suspected or confirmed by estimation of prothrombin activity vitamin K should be given (*see page 599*).

Rare complications.

Three rare complications of gastroduodenal bleeding are worth mentioning. Temporary and even permanent **blindness** may follow recurrent bleeding in an already very anæmic subject, and this is a powerful argument in favour of adequate blood transfusion. **Coronary thrombosis** may be precipitated by acute hæmorrhage and may cause diagnostic difficulties unless remembered. **Acute perforation** occurs soon after bleeding in 1 per cent. of cases. The diagnosis may be extremely difficult as the classical picture of perforation may be entirely lacking in an anoxæmic patient. Unusual tachycardia or abdominal distension should prompt examination for absence of liver dulness and peristaltic sounds, and an X-ray film to seek air under the diaphragm may be needed.

F. AVERY JONES.

CHAPTER VI

Medical Emergencies in Obstetrics and Gynæcology

THE urgent conditions which will be considered here are those of an obstetrical or gynæcological nature, which do not need immediate surgical or obstetrical intervention. Obstetrics and gynæcology form a regional branch of medicine, into which may intrude emergencies of a varied nature affecting all the main systems. The division between the physiology and pathology of pregnancy is often a fine one: it is thus necessary to look ahead in obstetric complications, since a condition may be better treated when it is potentially rather than actually urgent.

OBSTETRICS

ABDOMINAL PAIN DURING PREGNANCY

Abdominal pain of some degree is frequent in the pregnant woman. The manifestations of a definite surgical emergency, such as acute appendicitis, may be obscured by the pregnant uterus, and the emotional attitude of the patient may exaggerate the pain of more trivial lesions. Thus the diagnosis of abdominal pain assumes importance. Appendicitis and intestinal obstruction must be considered if the patient is vomiting. Cholecystitis, too, is not uncommon during pregnancy. It may be noted that pyrexia is not usually found with pain caused by some complication of the pregnant state itself, though a symptomless rise of temperature to 99° F. is not uncommon in the early months of pregnancy. Pain is not often associated with the vomiting of pregnancy.

EARLY PREGNANCY.

The common causes of abdominal pain in early pregnancy are:—

- Threatened abortion (*page 95*).
- Ectopic gestation.
- Stretching of the round ligaments.
- Stretching of the uterus.

Ectopic gestation.

In the early months ectopic gestation must always be considered as a possible cause of acute lower abdominal pain. It is one of the most frequently misdiagnosed acute abdominal conditions and, since it must be treated surgically, it is important to differentiate it from other causes of pain which might be treated medically. The typical history is of a short spell of amenorrhœa ("missed period"), followed by sudden severe colicky pain in the lower abdomen, usually one-sided. Vaginal bleeding of a varying degree occurs. Frequently there is a history of "one child sterility." The signs are a raised pulse rate with some degree of shock and hyperæsthesia of the lower abdominal wall with "guarding" rather than true rigidity. On vaginal examination there is exquisite tenderness in the pouch of Douglas and a tender mass may be felt there or in the lateral fornix.

In such a case the diagnosis may be very easy, but there are others which do not conform to the typical picture. These are usually cases where the pregnancy has formed a mole which has not been extruded from the tube. In general, the mass felt is very tender and, if still in the tube, is felt far out laterally towards the pelvic wall as distinct from the mass in salpingitis which is drawn down into the pouch of Douglas. If there has been any marked leakage of blood into the peritoneal cavity a useful accessory sign is the exquisite pain and tenderness elicited when the posterior fornix of the vagina is merely touched.

Stretching of the round ligaments.

Pain from this cause usually occurs about the twelfth to twentieth weeks of pregnancy. It is felt along the course of the ligament, i.e., along a line from the mid-point of the inguinal ligament to the side of the uterus and is more frequent on the right side. It is relieved by rest, and no special treatment is needed.

Stretching of the uterus.

This may cause indefinite pain at any stage of pregnancy. On abdominal examination the uterus is tender, and the patient states that she feels "tight" there. These cases are difficult to explain as some occur in the early months before hydramnios is likely. Relief may be obtained by giving progesterone 10 mg. daily for a week or so. (In later months, multiple pregnancy or

hydramnios may be responsible, calling for prompt obstetrical treatment).

THE LATTER HALF OF PREGNANCY.

The causes of abdominal pain at this stage of pregnancy are:—

Breech presentation.

Accidental hæmorrhage and placental separation.

Degeneration of a fibroid.

Pyelitis (*pages 58 and 272*).

Hæmatoma of the rectus muscle (*page 66*).

Breech presentation.

This commonly causes pain in the fundus over the foetal head. The pain is characteristic of the malpresentation and can be relieved by version.

Accidental hæmorrhage.

In this condition, pain is usually over the placental site, but there may be scattered areas of tenderness over the uterus caused by small hæmorrhages in its wall.

Treatment consists of rest, analgesics, and vitamin E (10 mg. three times a day) in the less serious cases. Appropriate obstetric treatment will be indicated in others.

If there is doubt as to the diagnosis of accidental hæmorrhage of the concealed type in an acutely ill patient, careful ascultation should be made for foetal heart sounds. If these can be heard there will be no question of serious accidental hæmorrhage.

Degeneration of a fibroid.

This may occur at any stage of pregnancy but is commoner in the latter half. The fibroid is felt as a tender, discrete swelling. Most cases settle down with rest and analgesics.

ABORTION AS A MEDICAL EMERGENCY

The treatment of abortion is expectant until it is inevitable and when it is inevitable it is to see that it is complete. The emergency of abortion is therefore firstly medical, but it becomes surgical if the products of conception are retained.

Patients threatening to abort are those known to be pregnant who have some vaginal loss without established pains or dilatation of the internal uterine os. Such patients should be put to bed at once and given a sedative such as Papaveretum B.P.C. 22

mg. (gr. $\frac{1}{3}$). This is preferable to morphine to which there may be idiosyncrasy.

The patient should be confined to bed for a week following the cessation of any loss of bright blood. During this time phenobarbitone 32 mg. (gr. $\frac{1}{2}$) should be given three times a day. There now appears to be no evidence of the efficacy of any endocrine preparations in the treatment of threatened abortion.

In all such cases the uterine position should be ascertained. If retroversion is present the patient should lie prone for half-hour periods frequently. The bladder should be emptied regularly and simple aperients used. Coitus should be forbidden till full placentation has taken place at four months.

Should abortion threaten at a rather later time than is usual, *e.g.*, at four or five months, the abdominal pain and tenderness over the uterine fundus may lead to mistakes in diagnosis, especially if the history of amenorrhœa is not obtained or is concealed. It should be remembered that "degenerating fibroids" are not common in young married women and that at this stage of pregnancy the uterus may be markedly cystic.

If the abortion is in the process of completing itself or is incomplete, hæmorrhage may become alarming and dangerous. Usually the uterus will have to be evacuated surgically, but bleeding may be arrested temporarily, and the abortion sometimes completed, by the use of oxytocics. Ergometrine should be given either intramuscularly or intravenously according to the severity of the bleeding. Intravenously a dose of 0.25 mg. should be injected, followed by another 0.25 mg. intramuscularly. By intramuscular injection alone 0.5 mg. may be given. Pituitrin or pituitary derivatives may be given in doses of 5 units intramuscularly, but as they may cause shock they should only be used when ergometrine is not available.

Grave emergencies may arise in abortion if blood loss is great or if there is shock. Both these conditions may arise, particularly if the abortion is self-induced. Blood loss demands replacement by transfusion, every care being taken to use cross-matched compatible blood, which should be Rh. negative if the Rh. grouping of the patient is not known. Not merely may transfusion accidents occur if Rh. positive blood is given to an Rh. negative patient, but the mother will also manufacture Rh. antibodies, which may result in any future pregnancy being com-

plicated by erythroblastosis (*see pages 47 and 588*). No Rh. negative female from puberty till the menopause should be given Rh. positive blood if it can possibly be avoided.

Should the patient have induced abortion by the use of soapy solutions injected with a Higginson's syringe, a state of marked shock may result. The patient collapses, has a thready or imperceptible pulse, a low blood pressure and may be cyanotic. Such patients are in grave danger—immediately from shock, and more remotely, from anuria. Immediate treatment consists of stimulation of the circulatory system by analeptics (*see page 608*) and oxygen (*see page 569*). Transfusion is usually necessary and may be difficult to carry out owing to the collapsed state of the veins (*see pages 17 and 586*). In view of the definite possibility of anuria supervening from a lower nephron lesion, the circulation should not be overloaded by too much fluid until an adequate urine output has been obtained (*see page 269*). In some areas great help can be obtained from the Emergency Obstetric Units ("Flying Squads"), which enable adequate treatment to be given at home, thus avoiding the risks of moving the patient when in a state of shock.

Medico-legal emergencies may also arise in cases of abortion (*see page 494*). If the state of the patient is dangerous, dying declarations may have to be obtained (*see page 492*), and knowledge that abortion has been induced may lay on the practitioner the obligation of notification to the Coroner or Medical Officer of Health.

Pregnancy tests.

It is sometimes important from the medico-legal point of view to be able to establish whether at any given time a patient was pregnant or not. The value of the evidence often depends on the time when it was obtained and so its collection is a relatively urgent matter.

For the establishment of a diagnosis of pregnancy, the biological tests (Aschheim-Zondek, Friedman, Hogben and male toad tests) are of great value, but must always be used in conjunction with the clinical findings. The A-Z test is probably the most accurate, but the Friedman and Hogben tests are easier to perform. In general, the tests become positive within about a fortnight of the first missed period, and become negative within a fortnight of delivery or abortion.

A positive A-Z test indicates pregnancy in 98 per cent. of cases (the exceptions being due to the menopause, some tumours, and minor degrees of thyroid imbalance), but a negative test does not carry such a high degree of accuracy. Negative Friedman tests may be wrong in 5 to 8 per cent. of cases.

In making a diagnosis between secondary amenorrhœa and pregnancy it is important to bear these facts in mind the more since a clinical diagnosis may be wrongly negative also in early pregnancy because the softness and flaccidity of the uterus may be wrongly interpreted.

Little help is gained by using pregnancy tests in cases of threatened abortion to determine whether or not the pregnancy is still continuing. Pregnancy tests depend on the production of hormones by the chorionic tissue. This may remain active, and the pregnancy tests positive, for 14 days or more after the foetus is dead. Similarly, if the production of the hormone by the chorionic epithelium is deficient, the test may be negative and lead one to believe wrongly that the pregnancy has come to grief. Should tests give results which seem at variance with clinical findings, it is best to repeat them after waiting a fortnight. In general, a negative or "doubtfully positive" test in a patient with a threatened abortion does not augur well for the continuation of the pregnancy as it points to possible abnormality of the chorionic tissue.

HYPEREMESIS GRAVIDARUM

Some nausea or vomiting is conceded as a symptom of pregnancy; hyperemesis is a condition in which the degree of vomiting is so exaggerated as to distress the patient or affect her general condition. Faced as yet with an unknown ætiology, treatment is mainly expectant, prophylactic of possible complications (dehydration, polyneuritis, and liver damage), and observational of the patient's general condition to determine whether termination of the pregnancy may be necessary.

Treatment.—In mild cases of hyperemesis the patient should be encouraged to continue her normal life reassured by the knowledge that the sickness is bound to diminish. The bowels should be made to act daily, and for several days meat and all fatty foods should be avoided. Glucose and sugary foods such as jam and honey should be taken freely. Antihistamine drugs (*page*

611) such as dimenhydrinate (Dramamine) 50 mg. help considerably.

Should improvement not occur, or if the vomiting is already affecting the general condition as indicated by a rise in pulse rate, dehydration, or ketonuria, the patient should be put to bed in hospital away from the worries of her home. From the onset of treatment there, careful records should be kept of the fluid intake and output, the pulse rate and temperature, the presence of ketone bodies and the amount of albumin in the urine, the blood pressure, and the general clinical state. If vomiting is only moderate, fluids by mouth should be restricted to 2 pints (approximately 1 litre) for 24 hours, and the diet should be of a carbohydrate type, supplemented by Marmite for its vitamin B content. Usually, on isolation, improvement occurs, and the diet may be gradually increased. Chlorpromazine (Largactil) is recommended (*see page 71*). Should vomiting be severe, however, the condition is serious and fluids must be given either by rectal drip or intravenously. Up to 6 pints (approximately 3 litres) daily of 5 per cent. glucose can be administered rectally. If necessary, saline with 5 per cent. dextrose,* 5 pints (approximately 2.5 litres) in 24 hours, may be given intravenously. Five units of insulin may be given after each pint of intravenous dextrose-saline. Sleep should be aided by hypnotics; an easy way is to give 1.3 to 2.0 G. (gr. 20 to 30) of chloral hydrate rectally with the saline. Calcium gluconate should also be given for the protective action which it is thought to have on the liver (10 ml. of a 10 per cent. solution intravenously on alternate days).

Aneurine hydrochloride, 25 mg., and pyridoxine, 50 mg., should be given daily by intramuscular injection. Some cases have responded to Injection of Suprarenal Cortex B.P.C. 20 ml. daily, intramuscularly. If medical treatment fails, the possibility of the termination of the pregnancy arises. This is necessary should delusions and polyneuritis (Korsakow's syndrome) appear (the aim of vitamin B treatment is to prevent this). Retinal hæmorrhages and exudates are also indications for speedy termination.

Excessive vomiting in late pregnancy may be a symptom of impending liver failure ("acute yellow atrophy") due usually

* This is sometimes ordered as "glucose" but dextrose should always be used intravenously.

to infective hepatitis complicated by a pregnancy. The onset is usually with epigastric pain and vomiting. Jaundice soon follows and the urine is scanty and bile stained. The pulse is deceptively slow. Progress is rapid. Coma or delirium supervene and death follows in severe cases. Diagnosis from phosphorus poisoning and eclampsia has to be considered. So grave is the condition that pregnancy must be terminated at once. Medical treatment is on the lines suggested for liver function failure (*page 82*).

Vomiting during delivery at term is apt to be complicated by peptic aspiration pneumonia (*see page 468*).

JAUNDICE IN PREGNANCY

Jaundice is one of the ills of the pregnant woman which may be easily and erroneously attributed to the pregnant state. Valuable time may be wasted before the causal condition unrelated to pregnancy is discovered (*see page 81*).

An example is gas gangrene infection which sometimes presents in pregnancy with jaundice when the foetus has died following induction of labour. It may also appear in the puerperium from a similar infection of a laceration. This infection makes the patient ill to a degree out of proportion to her local condition. The red cell count falls rapidly, jaundice appears and there is shock and peripheral circulatory failure. Swabs and blood culture show *B. Welchii* but an occasional anærobic organism without signs of acute infection may be a mere contaminant. Treatment should be by massive penicillin dosage (1 mega unit a day) and anti-gas gangrene serum 100,000 units intravenously at once and 50,000 units six hourly afterwards (*see page 579*).

ECLAMPSIA

Eclampsia is a grave emergency. The plethoric appearance of the patient, her pregnant state, and the free bubbling mucus at the mouth constitute a typical picture. It generally begins before or during labour and when it occurs in the puerperium the possibility of other causes of fits (*e.g.* cerebral tumour or epilepsy) must be seriously considered.

Management.—First treat the fits; next confirm the diagnosis by taking the blood pressure and testing the urine for albumin, and then start medical treatment based on the lines laid down by

Stroganoff. Most eclamptic patients are best nursed in hospital and their transfer should be arranged while they are under the influence of sedatives. The doctor should accompany his patient or call in the obstetric "Flying Squad."

Treatment during the fit.—As in all attention to the eclamptic patient, treatment should be as gentle and purposeful as possible. Roll her on to her right side with her head over the edge of the bed; keep the mouth open by a gag or wooden spoon; remove the mucus which pours out of the mouth and on no account let her head fall back or her throat will fill with mucus. When fits occur in rapid succession give thiopentone 0.25 to 0.5 G. intravenously. This is preferable to chloroform. If there is cyanosis, administer oxygen (*see page 569*). Should breathing fail, use artificial respiration (*see page 543*).

Treatment after the fit.—The patient should be put in a quiet room, darkened sufficiently to encourage rest, but not dark enough to impede the nurse. Continued careful observation of the patient, the blood pressure, the output of urine, and the amount of albumin, is needed. The importance of skilled nursing cannot be overstressed. The patient should be put between blankets and the foot of the bed raised to enable secretions to run away from the air passages. She should lie on her side, with her head turned to that side, the position being reversed every two hours to avoid hypostatic congestion of the lungs. Catheterisation should be performed and the catheter left in with a clip at its end. Urine can then be released four-hourly without disturbing the patient. This also enables the amount of albumin and the output of urine to be measured accurately. Sharp needles should be used for any necessary injections, which are best given at times coinciding with the maximum effects of the sedatives used. These precautions in technique are intended to reduce the risk of precipitating further fits.

Fluids (25 per cent. glucose in water is the best) may be given by mouth, very gently and in small quantities. If they are "pushed," further fits may be provoked.

Sedatives.—Some modification of Stroganoff's method is nearly always used, the basis being morphine and chloral. Morphine 16 mg. (gr. $\frac{1}{4}$) is given immediately and repeated if necessary after $\frac{1}{2}$ to 1 hour. Chloral hydrate 3 to 4 G. (gr. 45 to

60) is given rectally following the morphine. When given by mouth (in half the above dosage), chloral is frequently vomited, and repeated doses cause gastric irritation. This can often be avoided by diluting the dose to 60 ml. (2 fl. oz.) and flavouring with syrup of ginger. Subsequently, morphine and chloral are repeated according to need in amounts sufficient to keep the patient quietly resting. A total dose of 65 to 100 mg. (gr. 1 to 1½) of morphine and 16 G. (gr. 240) of chloral (rectally) is usually necessary in the first 24 hours. Alternatively, paraldehyde 5 ml. given intramuscularly (*see page 597*) may be used instead of chloral if no more fits occur after the first dose of morphine. It should not be repeated, however, more often than 12-hourly. It causes a prolonged sleep, and avoids ordering more frequent doses of other sedatives. Sedative treatment should be continued for 24 hours after the cessation of fits. In some rural areas where there may be delay in admitting a patient to hospital bromethol (Avertin, Bayer) may prove a useful sedative provided its use is agreed mutually by the doctor and hospital. 5 ml. are dissolved in 230 ml. of water (*see page 481*) at body temperature and given per rectum about half an hour before the ambulance arrives. The solution must be freshly made and tested by Congo red indicator to exclude acidity. If it turns blue the solution should be rejected. These inconveniences together with some difficulty in obtaining the substance limit its general use.

Treatment for special cases.—For patients who do not respond favourably to routine treatment or in whom coma supervenes, the use of sucrose, 500 ml. of a 10 per cent. solution intravenously, to reduce cerebral oedema may be considered. It also provides glycogen to protect the liver. If cyanosis is marked, oxygen should be administered and venesection should be performed.

Obstetrical treatment is merely mentioned here. When fits have ceased for 48 hours, labour should be induced. A few patients will need Cæsarian section.

ANURIA AND OLIGURIA IN THE OBSTETRIC PATIENT

Anuria in the obstetric patient may arise from lower nephron lesions in cases of abortion or accidental hæmorrhage (*page 95*); from toxæmia of pregnancy (*pages 98 and 100*); or from the use of sulphonamides without due precautions (*page 264*).

Anuria is in form either necrosis of the kidneys or from a lower nephron lesion in cases of abortion or accidental hæmorrhage requires special treatment. (See also page 262). The recent studies of Bywaters, Bull and their colleagues have shown that, in these circumstances, injury to the tubules is caused by myohæmoglobin or, to the cortex, by anoxia, and that recovery may be possible if the patient is tided over the phase during which uræmia may supervene. This type of anuria is diagnosed by the characteristic history of abortion, toxic hæmorrhage, or blood transfusion reaction, followed by the passage of a minimal quantity of urine, which may be blood-stained. During this phase of oliguria, it is dangerous to overload the circulation with fluid. Nitrogen metabolism is depressed by a special diet (see page 262).

POST-PARTUM HÆMORRHAGE

There is nowadays a more definite attitude towards the treatment of post-partum hæmorrhage. Obstetrically, under suitable conditions, manual removal of the placenta is more frequently advocated. Oxytocics are used more freely too, and every doctor should now carry ergometrine in his obstetric kit. In hospital, ergometrine 0.5 mg. intramuscularly is used prophylactically by many as a routine in instrumental deliveries, being administered when the anterior shoulder of the baby is born. When hæmorrhage occurs after delivery, ergometrine can be given irrespective of whether the placenta has been delivered or not. The dose is 0.25 mg. intravenously or 0.5 mg. intramuscularly and this controls the bleeding, though there is a risk of the placenta being retained by uterine contraction. As the bleeding ceases, appropriate steps can then be taken either for blood transfusion to be given, or for the patient to be prepared suitably for manual removal of the placenta if this is necessary. In post-partum hæmorrhage, the value of the Emergency Obstetric Units must also be mentioned.

ACUTE INFECTIONS OF THE GENITAL TRACT

Fulminating septicæmia and peritonitis demand emergency treatment before bacteriological examinations are completed. Penicillin must be given in large doses (up to 500,000 units three-hourly by intramuscular injection) and these doses continued till the clinical condition of the patient improves. Then the same

dose can be given six-hourly. This treatment will be indicated for most organisms likely to cause such infections. In infections arising from abortion, it is wise to give sulphonamides additionally, as coliform organisms are often associated with others (*see page 599*). If these are proved later to be present treatment should be changed to streptomycin 1 G. daily by intramuscular injection (*see page 605*).

Having initiated treatment, routine investigations must be made. A vaginal swab should be taken from the posterior fornix. The sensitivity of the organisms to antibiotics should be assessed. A catheter specimen of urine will also be needed and a full blood count and blood culture.

It is a legal obligation to report to the Medical Officer of Health any pyrexia in the puerperium which reaches 100·4°F. (38°C.) or more within 14 days after childbirth or miscarriage.

Certain emergencies which arise when pregnancy is complicated by an illness of medical nature will now be considered.

PREGNANCY AND HEART DISEASE

Pregnancy in a patient with heart disease constitutes a potential emergency because of the increased demands on the heart as pregnancy advances. Most of the patients suffering from heart disease during pregnancy have rheumatic mitral or aortic valvular lesions, but a small number of women with congenital lesions survive, marry and may conceive. In older patients and in those suffering from exophthalmic goitre, myocardial disease may be present.

The urgency of the situation depends on the extent of the lesion, and on the degree of cardiac compensation. In rheumatic cases, the length of time which has elapsed since the onset of acute rheumatic infection is important, a short time being less favourable than a longer interval. The economic status of the patient, too, is often a decisive factor, since it determines the amount of domestic help available.

Decompensation is, of course, the commonest complication, especially at about 30 weeks of pregnancy or in the puerperium; the importance of the stress of labour tends to be exaggerated. Decompensation is more likely to occur during the later than the earlier stages of pregnancy. It is not unusual to see sudden and

unexpected deterioration in a patient whose cardiac reserve seemed earlier to justify an excellent prognosis.

Treatment.—Prophylaxis is important in avoiding the full-blown emergency. A patient with a not fully compensated lesion should be told to conserve her energy carefully, avoiding stairs, and resting as much as possible. Respiratory infections and anæmia should be carefully treated.

During pregnancy the patient should be observed carefully for early signs of failure. Oedema is best sought in the bases of the lungs since, when it affects the ankles alone, it may be part of the picture of normal uncomplicated pregnancy. The treatment of decompensation is medical (*see page 163*).

Relatively few patients with heart disease give rise to anxiety during pregnancy and labour but induction of premature labour is occasionally necessary if dyspnœa increases, or the size of the uterus interferes with sleep. The emergency of having to terminate pregnancy by operative means may arise if decompensation becomes marked. If this is done, sterilisation should be effected at the same time.

Sudden cardiac failure sometimes occurs dramatically during labour, especially in the older multipara with myocardial disease. The child has usually just been born. Analeptics (*see page 608*) and oxygen should be administered, and an abdominal binder with a pad beneath it put on to compensate for the sudden alteration in the circulation consequent upon the evacuation of the uterus.

The outlook for patients suffering from congenital heart disease complicating pregnancy is much the same as for those with rheumatic valvular lesions with the same degree of compensation. Only the most fit patients, with minor lesions or an extraordinary degree of compensation, survive to an age when pregnancy is likely. Cyanosis is of serious prognostic significance.

A previously existing bacterial endocarditis may flare up during pregnancy, or if puerperal infection occurs.

PULMONARY TUBERCULOSIS AND PREGNANCY

Pulmonary tuberculosis complicating pregnancy renders the patient liable to some emergencies other than those which each condition alone might cause.

If emergency situations are to be avoided, full co-operation between the physician and the obstetrician is necessary, as there is a danger of each assuming that the other is doing the supervising.

Pregnancy does not appear to aggravate pulmonary tuberculosis or cause any specific emergencies, but rapid deterioration often follows parturition. The sudden emptying of the uterus leads to increased respiratory movements and a rapid spread of disease may occur.

Patients with pulmonary tuberculosis are often warned of the danger of pregnancy (but are not always instructed in how to avoid it). They therefore tend to become worried during pregnancy and the possibility of acute mental disturbance is always present.

ANÆMIA AND PREGNANCY

Anæmia, usually hypochromic, frequently complicates pregnancy and may constitute an emergency if its degree is such that the patient's condition would be jeopardised by loss of blood at delivery. Such anæmia is usually seen in older multiparæ whose hæmoglobin levels have been progressively falling before pregnancy.

If anæmia is diagnosed early in pregnancy, treatment should be on the usual lines by massive iron or other therapy (*page 171*). If, however, it is only discovered late, transfusion may be necessary, great care being taken to avoid transfusion accidents by careful cross-matching and the use of Rhesus-negative blood.

HYPERTHYROIDISM AND PREGNANCY

Hyperthyroidism is usually mild when pregnancy supervenes but even so, emergencies may arise. Thyroid crises should be dealt with as described on *page 248*. Myocardial failure is serious, and justifies the termination of pregnancy.

ADDISON'S DISEASE AND PREGNANCY

Crises in early pregnancy from vomiting and in late pregnancy and labour from adrenal insufficiency should be dealt with as described on *page 253 et seq.*

URGENT SKIN CONDITIONS ASSOCIATED WITH PREGNANCY (*see page 430*)

PASSIVE IMMUNISATION OF PREGNANT WOMEN (*see page 321*)

PREGNANCY IN THE DIABETIC

THE MOTHER.—The emergencies which may arise are:—

- (1) Ketosis, particularly in early pregnancy because of the metabolic changes taking place.
- (2) Hypoglycæmia may occur, particularly towards the end of pregnancy and is attributed to lowering of the renal threshold which allows excessive leakage of sugar from the blood to the urine. Probably it is the occasional *sudden* and transient fall of the renal threshold that accounts for symptoms such as "fainting attacks" of hypoglycæmic origin. In diabetes theoretical considerations do not always apply to individual cases, and it is wise to be guided by the blood sugar levels.

With careful management and full co-operation between the patient and her physician and obstetrician both of these emergencies may be avoided, and if they occur they should be treated on the lines laid down in Chapter XIII.

- (3) Toxæmia. There seems to be an increased risk of pregnancy toxæmia and the emergencies of eclampsia may have to be dealt with.

During labour the usual diet should be continued as long as the patient can take it, and then 20 G. of glucose given every two hours, with an extra 30 G. at the onset of the second stage. The risk of hypoglycæmia increases during labour because of muscular action, and so insulin should be cut down until it is over. Half a unit should be given for each gramme of glucose retained.

THE BABY.—Many of the emergencies confronting the baby are obstetrical in nature from the fact that he is often heavy and immature. Hydramnios is frequently present also.

Neonatal death from hypoglycæmia due to over activity of the pancreas has occurred, and the care of the diabetic's newborn child should always have the priority of a potential medical emergency. Glucose should be given until breast feeding is established (*see page 286*). The state of the baby is the best guide to the dose but occasionally a blood sugar estimation may be indicated.

Premature death of a foetus in utero has been thought to be more likely in those patients who develop hyperprolanaemia. An attempt to avoid this risk can be made by suppressing anterior pituitary activity with large doses of oestrogens. A daily dosage of 25 to 40 mg. of stilboestrol by mouth, or 150,000 units of oestradiol benzoate by intramuscular injection is needed from about the fifth to seventh months of pregnancy. This treatment is expensive and protracted. It would be best to confine its use to patients who have a history of foetal death in utero, or in whom estimation of blood gonadotropins has been made.

VARIOUS ACUTE EMERGENCIES IN PREGNANCY

Syncope.

A faint may easily occur in the apprehensive patient, particularly if some obstetrical manipulation such as external version is being performed. The head should be lowered and the feet raised. A simple stimulant (sal volatile, brandy or smelling salts) may be used.

Embolism.

Embolism by air or clots may cause sudden emergencies during pregnancy and should be dealt with as indicated on *pages 30 and 133*. Very rarely pulmonary embolism may be caused by amniotic fluid entering the maternal circulation. The condition arises during labour and the puerperium and is characterised by the sudden onset of cyanosis, dyspnoea and shock. No treatment is possible other than restorative measures. Most cases have proved fatal but if the patient shows signs of recovery it is important to treat the post-partum hæmorrhage which frequently occurs.

Acquired afibrinogenæmia.

In patients who have recovered from the acute effects of amniotic fluid embolism, in cases of accidental ante-partum hæmorrhage and in some cases of foetal death in which the foetus is long retained, a condition of afibrinogenæmia may arise. It is produced by intravascular coagulation or reduction by a fibrinolysin of circulating fibrinogen which the liver cannot replace quickly enough.

Hæmorrhage occurs from any incision or raw surface and is very difficult to stop. In many cases blood transfusion will supply the necessary fibrinogen but, particularly in accidental hæmorrhage, this may not be enough. Human fibrinogen may have to be given in addition. Tests must be made to determine absence or deficiency of clotting in a sample of freshly drawn venous blood. According to Wiener a deficient clot dissolves within an hour if incubated at 37° C. Should this dissolution not occur there is no necessity to give fibrinogen. This simple test is much quicker than laboratory methods to determine the fibrinogen level in the blood (normally 190 to 330 mg. per 100 ml. with a minimum level of 100 mg. to ensure hæmostasis). There is doubt about the correct amount of fibrinogen to administer. Amounts from 500 mg. to 12 G. have been quoted. Probably from 4 to 6 G. are necessary (obtainable from The Lister Institute, Chelsea Bridge Road, S.W.1. Tel.: SLOane 2181). Failing fibrinogen quadruple strength plasma could be used; 570 ml. (1 pint) yields about 4.4 G. of fibrinogen.

Fractured ribs.

Consequent upon coughing or the stress of bearing down during delivery, it may happen on rare occasions that a pregnant woman fractures a rib or the sternum or even dislocates one or more ribs. There will be sudden pain and local tenderness. Treatment consists in simple immobilisation by strapping. The same stresses may cause subcutaneous and mediastinal emphysema (*see pages 25 and 140*).

Suprarenal hæmorrhage.

Very rarely suprarenal hæmorrhage may cause acute abdominal symptoms or sudden collapse in the later weeks of pregnancy. Usually there is some preceding complication such as toxæmia, abortion, vomiting or hæmorrhage (*see also page 253*).

Thrombocytopenic Purpura.

The pregnant patient who suffers from chronic thrombocytopenic purpura is prone to hæmorrhage both antenatally if there is toxæmia and after delivery. A blood transfusion may therefore be given prophylactically near term.

EMERGENCIES IN ROMAN CATHOLIC MOTHERS DURING PREGNANCY

In grave illness during pregnancy, when the foetus is viable (*i.e.*, of six months' gestation) and vaginal delivery dangerous, Cæsarian section is permitted when it offers the only chance of saving the mother's life. It should not be done if it does not offer any chance of saving her life. In such a case, death should be awaited and no pressure should be brought to bear on a mother to make her provide by surgery for the baptism of her child. If uterine baptism (*see page 507*) is possible, it should be undertaken.

If a mother dies in pregnancy, the Code of Canon Law 1918 states that Cæsarian section should be done in order that the foetus may be baptised and this applies whether uterine baptism was given or not. There would be no obligation to do this if there were good reasons to believe that the foetus was dead. When death is not sudden, preparation for post-mortem section and baptism should be made.

POST-MORTEM DELIVERY

When a pregnant woman dies suddenly or from some foreseen cause the problem of delivery becomes urgent. The act of dying may precipitate labour and should the head be found on the perineum a forceps delivery may avoid the repugnant procedure of post-mortem section.

When death is not sudden the baby will usually predecease the mother. When the mother dies first the baby is not likely to survive long but may live for 20 minutes after the sudden death of its mother.

In such circumstances a classical Cæsarian section should be speedily performed when the mother's respiration and circulation have ceased. Permission of the husband need not be awaited. Incisions should be closed with due care as there are records of recovery of a supposedly dead mother.

PSYCHOLOGICAL EMERGENCIES IN PREGNANCY AND THE PUERPERIUM

The many psychological and physical changes during pregnancy make it understandable that psychological disturbances should occur. Fortunately, few are emergencies in the strictly medical

sense, but many appear urgent to the woman herself. Worries about possible foetal abnormality, conflict about an unwanted pregnancy, and fear of labour will need proper care and explanation. These minor troubles tend to conceal the more serious ones arising from psychosis; the vocal hysteroid personality demands superficially more attention than does the more quiet and secretive obsessional. Thus, if careful observation is not made, and even if it is, a sudden psychiatric emergency may arise which may be fraught with tragedy.

During early pregnancy, neurotic manifestations are commoner than psychotic ones. Hysterical reaction to an unwelcome pregnancy may be associated with demands that the pregnancy be terminated and with threats or gestures of suicide. Termination on psychological grounds is seldom indicated and needs very careful discussion indeed with the psychiatrist. It may have to be considered in the psychotic when there is a history of obsessions, delusions, or depression either independent of pregnancy or recurrent with successive pregnancies. Urgent observation in hospital is necessary before deciding the course of action. Fortunately, the modern methods of treatment, notably electroplexy, are compatible with continuation of pregnancy and do not affect the baby. Termination, although altering the immediate situation, has its own psychological effect which may not be beneficial in the long run because feelings of guilt may be induced. Moreover, the psychosis may continue or even worsen, irrespective of abortion, particularly if this is complicated.

Psychosis in pregnancy is more common in the latter months or during the puerperium. There is a possibility of suicide or killing the baby, so that recognition and treatment is extremely urgent. First, it may be noted that obstetric stress such as continued pyrexia, eclampsia, and lactational difficulties help to precipitate a psychosis. Secondly, attention must be paid to important symptoms known to be associated with psychosis—continued sleeplessness, sudden fantastic remarks or confusion. The recognition of these, followed by prompt treatment may avoid catastrophe.

When the diagnosis of psychosis has been considered or made psychiatric opinion should at once be sought. The baby should be kept safely from the mother and weaned; the mother should be most carefully nursed and watched, and simple obvious means of suicide removed.

GYNÆCOLOGY

Gynæcological emergencies usually present as abdominal pain, or with some other marked symptom such as menorrhagia. Accurate diagnosis is important as many patients will need surgical treatment either immediately or when the emergency has been tided over. Conditions which justify medical treatment are the symptoms of menorrhagia and of the psychological states associated with amenorrhœa and pruritus; the inflammatory lesions of salpingitis; and certain causes of abdominal pain in women, *e.g.*, Mittelschmerz.

MENORRHAGIA

Menorrhagia is merely a symptom, and therefore its cause must be sought in all cases. As this may be in the genital tract (fibroids or pelvic infection), vaginal examination must always be performed. It is often difficult to diagnose with certainty a small submucous fibroid causing menorrhagia, as the uterus may only feel a little bulky and have no obvious deformity of outline. Exploration with a sound under anæsthetic may be necessary.

In many cases menorrhagia is caused by the gynæcological endocrine make-up of the patient, or by some general condition such as hypothyroidism, leukæmia or anæmia. Minor degrees of alcoholism, particularly if spirits are taken, may also cause excessive loss.

Menorrhagia due to essential functional causes may occur at puberty, during adult life (cyclic or acyclical bleeding), or at the menopause. Diagnosis is made by excluding general causes and local pelvic conditions, by a consideration of the history and type of loss, and, if necessary, by endometrial biopsy.

Although most patients will complain of the excessive loss, some, especially if the periods have always been heavy, will notice only the secondary effects of anæmia. Such cases tend to occur near middle life when the bone marrow response to hæmorrhage is waning.

Medical treatment may be necessary during the acute attack to combat a known cause, to correct resulting anæmia, and to prepare a patient for surgery.

EMERGENCY SYMPTOMATIC MEASURES.—The patient complains of a sudden drenching loss of blood and perhaps faints. She should be put to bed and examined to exclude any cause which calls for surgical treatment. Ergotamine tartrate, 1 mg., should be

given three times a day by mouth, or pitocin 0.5 ml. every four to six hours subcutaneously.

If the patient is a young girl (puberty menorrhagia), or if the history in an adult is suggestive of metropathia hæmorrhagica (i.e., spells of irregular excessive loss following short periods of amenorrhœa), help may be obtained from progesterone 10 mg. intramuscularly every day.

When gross anæmia is present a blood transfusion should be carried out. In all cases it is essential to treat the anæmia by giving iron by mouth (Compound Ferrous Sulphate Tablets, B.P.C. gr. 3, four times daily after food) or by injection.

Emergency treatment of the medical causes of menorrhagia.—Thyroid 32 mg. (gr. $\frac{1}{2}$) three times a day should be given if there is any evidence of hypothyroidism. Acute menorrhagia may be the presenting symptom in leukæmia, aplastic anæmia and purpura and emergency treatment for these diseases may be necessary (Chapter IX).

Puberty menorrhagia is usually a self-limiting disease which corrects itself if given time, a high protein diet, vitamins, iron and calcium. If the loss is severe, relief may be obtained by progesterone 10 mg. intramuscularly three times a week for the latter two weeks of the menstrual cycle.

In the adult patient, acute loss of blood at a period may be menorrhagia if the periods have been regularly spaced, or due to metropathia hæmorrhagica, in which case periods of amenorrhœa alternate with heavy losses at irregular intervals. Simple cyclical menorrhagia if needing endocrine treatment is best managed by giving 5 mg. methyl testosterone orally twice daily for 8 to 12 weeks. Flooding due to metropathia hæmorrhagica can be quickly checked by giving large doses of œstrogens, either stilboestrol 5 mg. or ethinyl œstradiol 0.25 mg. every two hours until the loss is reduced. Then either stilboestrol 2 mg. three times a day or ethinyl œstradiol 0.25 mg. once daily for a week. Following this a modified period will occur a few days later.

In patients nearing the menopause most endocrine preparations are of little help, as the ovaries are growing insensitive to stimuli. More often an artificial menopause is indicated after careful exclusion of malignant disease.

Emergency medical measures preparatory to operation.—Patients whose hæmoglobin is less than 50 per cent. are best

tided over by medical treatment. After acute blood loss, transfusion may be necessary, but is best reserved until just before operation. In some cases the temporary use of testosterone will enable anæmia to be combated.

PSYCHOLOGICAL DISORDERS IN THE GYNÆCOLOGICAL PATIENT

It is not surprising that symptoms referred to the sex organs may originate from psychological conditions. An anxiety state may have as its main symptom **amenorrhœa** of recent onset which may be due to fear of an undesired pregnancy or worry that the menopause is starting early. Assurance alone may not convince the patient and in these cases having excluded the possibility of a pregnancy, relief may be dramatic if a "period" is induced. Prostigmin 0.5 to 2 mg. subcutaneously should be given on three consecutive days. This will usually cause a loss from uterine congestion.

In the obsessional patient, an acute condition of **pruritus vulvæ** may occur and the woman may extensively traumatise the skin by scratching. The vicious circle may be broken temporarily by anæsthetising the area by the subcutaneous injection of Proctocaine—a proprietary preparation of procaine and butyl p-amino benzoate in almond oil.

Two small wheals are raised in the labia majora with 2 per cent. procaine, the site chosen being midway between the clitoris anteriorly, and the anus posteriorly. Through these areas, Proctocaine is injected in a fanwise manner with a long needle, first directing it forwards towards the clitoris, and leaving about 2.5 ml. of the solution along the course of the needle as it is slowly withdrawn. The needle is then swung round towards the anus and a further 2.5 ml. injected into the posterior half of the field. Thus, about 10 to 12 ml. in all are necessary. Usually relief can be obtained for a fortnight and this may break the circle or enable other treatment to be given. Proctocaine must not be used in the presence of infection.

EMERGENCY CONDITIONS OF INFLAMMATORY ORIGIN

Most cases of salpingitis, whether acute (due to the gonococcus or non-specific organisms) or chronic (due to the tubercle bacillus), are treated by non-surgical methods unless there are definite

reasons such as doubt in diagnosis or spreading peritonitis to indicate the need for surgical interference.

Diagnosis.—The differentiation of unilateral salpingitis from right-sided appendicitis is important, and particularly if the appendix lies in the pelvis. Helpful points are:—

- (a) The temperature—pulse ratio. In salpingitis the pulse rate is proportional to the pyrexia, whereas in appendicitis it is disproportionately rapid.
- (b) Rovsing's sign. Pressure is applied over the descending colon. In appendicitis pain is felt in the right iliac fossa when the pressure is released.
- (c) Headache is more frequently present in salpingitis than in appendicitis.

Treatment.—The patient is put to bed in Fowler's position and pain relieved by heat applied to the abdomen. Compound Tablets of Codeine B.P. (two every four hours), often suffice to relieve the pain, but pethidine (100 mg. intramuscularly), or even morphine 16 mg. (gr. $\frac{1}{4}$) subcutaneously, may be required. Chemotherapy (by a sulphonamide or antibiotic) should be used.

Drainage may be aided by the vaginal application of tampons of ichthammol B.P. (5 to 10 per cent.) in glycerin.

The social and medico-legal implications of salpingitis must be remembered. If gonococcal, the source of infection has to be traced, and obviously the husband will have to be examined. There is danger too, of infecting children, and this may make isolation important. If salpingitis originates from an abortion, the possibility of criminal interference must be considered (page 494).

VULVO-VAGINITIS IN CHILDREN

Owing to its contagiousness, this demands urgent treatment. Swabs should be taken to identify the causative organism and the patient isolated. All contacts should be examined. Sulphadimidine (Sulphamezathine) should be given in dosages proportional to the age of the child, the smallest infant receiving up to 3 grammes in the first 24 hours.

Penicillin in cases of gonococcal origin is valuable, 1,000,000 units being given intramuscularly during 24 hours. Cases not of gonococcal origin do not respond so well to penicillin. In view of

the possibility of mixed infections masking the gonococcus, it is probably wisest to give it in all cases.

Apart from baths and subsequent thorough drying of the area, local treatment is not usually necessary. In resistant cases, oestrogenic preparations may help (*e.g.*, stilboestrol 0.5 mg. twice daily).

ACUTE LOWER ABDOMINAL PAIN IN WOMEN

Acute lower abdominal pain in women may have many medical causes as well as being caused by lesions of the genital organs themselves. The latter cause pain in various ways. The dull heavy ache of an ovarian cyst or fibroid is caused by its weight causing stretching of tissues or by congestion of its pedicle. The lancinating pain of a tubal mole is of the nature of colic. The premenstrual pain of endometriosis is due to tension. Peritoneal irritation causes pain from inflammatory lesions or extravasated blood, and extension of malignant disease into nerves may cause severe pain.

Many patients need surgical treatment, and only a few notes on diagnosis and medical treatment are here relevant. Ectopic gestation must be excluded in women of child-bearing age who complain of marked unilateral pain and have a history of recent menstrual irregularity. In patients over 40, ill-defined lower abdominal pain may well be a symptom of carcinoma of the ovary, diverticulitis, or carcinoma of the pelvic colon with pericolic abscess. The last two conditions present a tender mass in the left lateral and posterior fornices which may easily be confused with an atypical tubal or ovarian swelling if their possibility is not remembered.

Degeneration of a fibroid may cause acute pain and tenderness. Symptoms usually settle with rest and analgesics.

MITTELSCHMERZ

In younger women, pain at the time of ovulation is not infrequent (*Mittelschmerz*) (*see also page 60*). Whilst usually a dull ache, it may be more acute if accompanied by free bleeding into the peritoneal cavity. In such cases the diagnosis of appendicitis is often made, and an unnecessary appendicectomy performed. The timing of the attack is typical (twelfth to fifteenth days of the menstrual cycle). There is often marked

hyperæsthesia on moving the cervix, and in the pouch of Douglas, from accumulation of blood there. A leucocyte count fails to reveal the leucocytosis so typical of a suppurative lesion. Treatment consists of rest in bed and the administration of analgesics. Operation is needed only if the diagnosis is in doubt, or more rarely if marked intra-peritoneal bleeding is present sufficient to raise the pulse rate to 120 or to cause pain not relieved by rest.

PAIN IN MALIGNANT DISEASE

Advanced malignant disease may cause urgent pain from nerve involvement. Intrathecal injection of absolute alcohol often gives marked relief, but in view of the dangers, this method should be used only in this type of case.

The technique is as follows. The patient lies with the painful side uppermost. The pelvis is raised by a pad so that the sacral and lumbar regions are elevated. The head is lowered and the body turned ventrally so that the posterior sensory nerve roots are uppermost. The skin is cleaned and thecal puncture performed in the fourth lumbar interspace. When C.S.F. is seen to escape, 0.5 ml. of absolute alcohol is injected very slowly, drop by drop, taking two minutes to do this. No attempt is made to mix alcohol with C.S.F. in the syringe. The patient is kept on her side for two hours. She will complain of numbness which disappears spontaneously.

KENNETH BOWES.

CHAPTER VII

Respiratory Emergencies

(For Acute Post-operative Pulmonary Complications, see page 459)

THE chief disorders of the respiratory system which are likely to call for urgent treatment are those in which respiration ceases or becomes painful or difficult, or in which cyanosis occurs or blood is coughed up.

CYANOSIS

Cyanosis is rarely in itself responsible for an urgent call, but rather the associated symptoms. Its appearance depends on the amount of reduced hæmoglobin in the blood. If this is 5 G. per 100 ml. or more, cyanosis appears. It may occur independently of respiratory failure as in some cases of congenital heart disease when venous blood crosses in the heart to the arterial side. In polycythæmia, cyanosis occurs because the total hæmoglobin is so high that even with perfect pulmonary ventilation there are always at least 5 G. per cent. of reduced hæmoglobin. Conversely, in severe anæmia with hæmoglobin less than 5 G. per cent., cyanosis is impossible.

The explanation of cyanosis in pneumonia and other lung diseases often presents difficulty. It does not follow that cyanosis will develop because a whole lung is out of action, as in complete collapse; the collapsed lung is nourished via the bronchial arteries, and the blood flow through it from the pulmonary artery to vein is practically abolished. Thus, there is no unoxygenated blood flowing back to the left auricle, and therefore no cyanosis.

In lobar pneumonia, cyanosis occurs because there is some blood flow through the solid lung. But oxygen will not alleviate it because oxygen cannot reach the blood via the solid lung, and the blood flowing through the healthy lung is completely saturated with oxygen.

In broncho-pneumonia, with smaller scattered lesions, the problem is different. Here cyanosis is caused by deficient ventilation of the lungs, but aeration of blood in the unaffected parts is normal. Oxygen will relieve cyanosis in this type of case.

As many cases fall between these two extremes it is wise to give oxygen in all cases.

It should be noted that cyanosis does not of itself cause dyspnœa, and that the effect of oxygen on cyanosis is variable. Generally speaking, cyanosis due to arterial anoxæmia of pulmonary origin will be relieved by oxygen, and especially when pneumonia is the main factor, whereas cyanosis of circulatory and chemical origin is unaffected.

DYSPNŒA

It is now realised that everyone's breathing is of a rate and depth which is most economical in terms of respiratory work. When the extra effort of getting air into the lungs (as in cardiac failure) or out of the lungs (as in asthma) is above a certain threshold then the patient is aware of it. This is dyspnœa or "breathing with conscious effort" (Samuel Gee). Increased pulmonary ventilation unassociated with any sense of difficulty is called hyperpnœa. The exact mechanism of adjustment of respiratory work is unknown but it is probable that the Hering-Breuer reflex (inhibition of inspiration by pulmonary distension) plays a part in it. When the lungs are stiffened expiration is initiated earlier. This causes shallow breathing and if the need for oxygen is increased the breathing is rapid too.

The various clinical conditions causing urgent respiratory embarrassment will now be considered.

ELECTROCUTION

The effects of an electric current on the body depend not only upon its voltage but also on the duration of the shock and the circumstances affecting conduction. The most important effects from the emergency point of view are on the nervous system—coma and bulbar palsy. Burns and fractures may also occur. The patient may also be unconscious, pulseless, and apnœic and appear to be dead.

Treatment.—Switch off the current. If this would take too long pull the victim away or push him away with a broom-stick. Pad the hands with dry clothing or wear gloves while doing this. If found in contact with or near a high voltage cable or rail call the engineer; otherwise there may be two victims instead of one.

Carry out artificial respiration (for method and duration see page 543). Give leptazol, 2 ml. intramuscularly repeatedly, and apply warmth. If the patient remains comatose but is breathing, or if breathing does not establish itself, lumbar puncture, and removal of 5 to 10 ml. cerebro-spinal fluid is advisable.

LIGHTNING

Although the majority of people struck by lightning recover unharmed, some are killed, and so a severe thunderstorm may be dangerous. During the storm it is best to stay in the house with doors and windows closed. Keep away from the fireplace, the main electric switch, and the wireless aerial. The telephone is safe to use, being fitted with lightning arresters and fuses at both ends.

If out of doors, take refuge in a substantial building or closed car but keep away from crowds. Avoid solitary trees, walls, wire fences, ponds and river banks. It is probably best, even if uncomfortable, to lie down in a ditch till the storm passes. It is safer to be thoroughly soaked than to be dry, since wet clothing may short-circuit most of the current. A person struck by lightning falls unconscious at once. He does not see the flash.

Treatment.—Artificial respiration should be started immediately. It may have to go on for several hours (see page 554) and so transference to a cabinet respirator should be considered. Do not assume that the patient is dead because the heart is inaudible. In these cases it can tick over undetectably. An E.C.G. may help to decide. Temporary flaccid paralysis of the legs is common and requires no special treatment. Acute hysteria may demand emergency measures (page 203).

HANGING

Death from hanging usually results from asphyxia. The heart continues to beat for some minutes after respiration ceases and it is wise to try artificial respiration, particularly if the person is found within five minutes of suspension. Recovery will be precluded if, as in judicial hanging, there has been a "drop," because this dislocates the cervical vertebræ.

DROWNING

In fresh water drowning enormous amounts of water are quickly absorbed from the lungs. Great hæmo-dilution occurs but the release of potassium by hæmolysis causes the K/Na ratio to rise. Death from ventricular fibrillation quickly follows.

In salt water drowning the inhaled fluid has a higher electrolyte concentration than plasma and so fluid passes into the lungs causing oedema. There is no hæmolysis and no disturbance of the K/Na ratio. Ventricular fibrillation does not occur. Respiratory failure comes on just before irreversible circulatory failure from anoxia causes death.

All deaths in water are not due to drowning. Sudden shock or "vagal inhibition" from stimulation of the larynx may be responsible. Fish stings (*page 471*) and sudden painful injuries such as a prolapsed intervertebral disc may play a part.

Treatment.—Artificial respiration by the Holger Neilsen method (*page 544*) must be started as soon as the victim is landed or in the boat. *Literally not one second must be wasted.* It must go on without interruption for at least 15 minutes and only then should any attention be paid (preferably by another person) to such measures as loosening clothes, draining the lungs, listening to the heart and so on. A few breaths in the first few seconds will do more than pure oxygen through a perfect airway could do 10 seconds later.

When recovery is occurring the movements of artificial respiration must be timed to coincide with the patient's breathing. Watch must be kept for recurrence of respiratory failure. Excessive stimulation may be dangerous. Pure oxygen should not be given except during artificial respiration. When breathing returns the victim should be turned on his side to lessen the work of breathing. The right side should be uppermost as this position lessens the risk of regurgitation. As soon as possible the patient should be removed to hospital.

When a person recovered from the water is obviously dead and an autopsy is held two important specimens should be obtained before they are spoiled. There are (1) peripheral lung and stomach contents for algæ and (2) blood from the right and left ventricles to show differences in electrolyte concentrations.

“WINDING”

This is usually a footballer's injury when a blow in the solar plexus causes inhibition of respiration, fall of blood pressure and perhaps loss of consciousness. Recovery has usually occurred before a doctor is called, but if it is slow the limbs should be raised and massaged towards the heart. Atropine 0·6 mg. (gr. $\frac{1}{100}$) should be injected and failing this Nikethamide 2 ml.

More often a blow on the lower chest causes spasm of the diaphragm and inability to breathe properly so the victim struggles to get air. If normal breathing is not quickly established artificial respiration should be used.

ACUTE LARYNGEAL OBSTRUCTION

(See also pages 310 and 435)

This state may be reached suddenly as when a large foreign body becomes impacted in the larynx, or a small one causes intense spasm of the vocal cords. When it is more gradual there are warnings of its onset, such as hoarseness and a feeling of phlegm in the throat which coughing does not dispel. Stridor and dyspnoea next appear and the patient becomes restless. Cyanosis is a late and dangerous sign. If acute symptoms following the inspiration of a foreign body are associated with a normal voice it is likely that the foreign body has passed the larynx.

In some people the larynx is amazingly insensitive. If a foreign body, even a large denture, disappears from the mouth, particularly during sleep, without causing choking, it does not necessarily follow that it has been swallowed rather than inhaled. It may be in a bronchus, so creating an emergency situation albeit a less urgent one.

In addition to a foreign body there are other causes of acute laryngeal obstruction. These are :—

1. Acute oedema of the larynx, which may complicate quinsy, septic pneumonia, and trauma by a sharp foreign body. It may also be an allergic or anaphylactic reaction.
2. Retropharyngeal abscess, which overlaps the larynx (*page 296*).
3. Diphtheritic (*page 310*) and streptococcal laryngitis.

In rare cases following explosion the epiglottis has been impacted in the glottis, thereby obstructing it and preventing artificial respiration. Digital disimpaction is essential.

Treatment.—The importance of a clear airway cannot be overestimated. I have seen a morphinised patient become moribund from respiratory failure and recover dramatically when the tongue was pulled forward.

Sometimes the initial choking attack subsides, but we must not too readily assume that all is well. The foreign body may have passed down to a smaller bronchus. The situation is still fraught with danger, and the patient should be X-rayed and bronchoscoped. Some of the extreme dyspnoea of apparent laryngeal obstruction may result from accompanying mediastinal emphysema (*page 132*) and so X-ray examination of the chest before tracheotomy is sometimes a wise preliminary step.

Where a foreign body is impacted in the larynx, the patient, usually a child, may be inverted and an attempt made to dislodge it with the finger. If not immediately successful the attempt should be abandoned, for it may cause dangerous spasm of the cords. An emergency tracheotomy should be performed (*page 569*).

When laryngeal obstruction is more gradual and gives warning of asphyxia preparations can be made for a planned tracheotomy. These should be made early to avoid Chevalier Jackson's criticism—"We always preach early tracheotomy but practically always do it late—dangerously late." The patient should be moved into or near the operating theatre and the surgeon called.

ACUTE LARYNGO-TRACHEO-BRONCHITIS

Inflammatory obstruction of the larynx and below may complicate respiratory infections and especially those of influenza and measles. It can follow endo-tracheal intubation. The sub-glottic area is the narrowest part of a child's larynx and its loose tissues readily swell. Gummy exudate and oedematous supra-glottic tissues may suddenly obstruct the larynx from above and make partial asphyxia complete. These cases are therefore always potential emergencies. Increasing restlessness often means that the acute emergency is not far off. No one who has seen one of these patients literally fighting for breath will ever forget it. The fact that the voice is often normal does not mean that the danger is less.

DIAGNOSIS.—This usually presents little difficulty but the other causes of "croupy" breathing such as foreign body, diphtheria and retropharyngeal abscess must be remembered.

Treatment.—The patient should be in an oxygen tent so that water vapour from a steam kettle and an aerosol of a surface tension-lowering detergent, *e.g.*, Alevaire, Bayer or trypsin (Trypure, Novo) can be given also. (Protect the eyes from the aerosol). (For aerosol machines *see page 604*). In desperate cases the lessened work of inhaling 20 per cent. oxygen in helium may tide the patient over a crisis. As the organisms are often penicillin resistant chloramphenicol (*page 606*) or tetracycline (*page 607*) should be used. If the condition worsens tracheotomy (*page 569*) should be done (preferably after laryngoscopy has confirmed laryngeal obstruction) even though bronchiolitis and pneumonia are present also. It is even worth while in a moribund patient as artificial respiration may then rescue him from death. The complications of mediastinal and surgical emphysema (*page 140*) may have to be faced.

EPISTAXIS

Epistaxis is only likely to occasion an emergency call when it is profuse or the patient is already very ill from some underlying cause.

HOME REMEDIES.—These are:—applying cold water to the nose; sitting upright with the feet in hot water and pinching the nostrils (preferably with a spring clip or clothes peg after putting vaselined wool into the nostril). These measures will usually have been tried before the doctor is called.

POSTURE.—Epistaxis usually arises from veins on the septum or in grooves on the turbinate bones. Negative pressure on swallowing dislodges clots which form on the free sides of the veins. Prevention of swallowing by allowing blood to drip out through the nostrils will allow clots to become adherent and so stop the bleeding. The next step should therefore be to give bleeding a chance to stop spontaneously by adopting the postural treatment described by Trotter. "Prop the patient well up with a comfortable inclination to one side; arrange a large pad of wool for him to dribble into; put a dental prop between his teeth; forbid him to breathe through the nose or swallow and give a substantial dose of morphia. Only by mouth breathing and a complete cessation

of swallowing can the bleeding area be given the necessary and, almost infallibly hæmostatic rest."

CAUTERISATION.—If the above measures fail the nose should be cleared by blowing and anæsthetised by applying cotton wool soaked in a 2 per cent. solution of amethocaine in 1 in 2,000 adrenaline hydrochloride. It can then be cleaned out with swabs soaked in weak hydrogen peroxide and inspected. If the bleeding point can be seen (usually at the front of the septum) it can be sealed by cauterisation.

Using a dental syringe anæsthetise the skin of the nostril and the mucous membrane of the septum with 2 per cent. procaine. Touch the bleeding point with an electric cautery at cherry red heat. If too hot it will cut the vessel and if too cold it will stick to it and tear it. Cauterisation by a silver nitrate stick is less frightening than the cautery for children.

PACKING.—When bleeding is profuse and its source cannot be seen the nose should be packed with half-inch gauze wrung out in the amethocaine and adrenaline solution. After about five minutes this should be removed and replaced by one inch iodoform gauze, or by Calgitex alginate gauze which has a hæmostatic action. The free ends of gauze should be kept in front and the nose packed from below upwards.

In the rare cases of persistent bleeding from vessels on the lateral wall and posterior part of the nose the post-choanal pack may be needed in addition to packing from in front. Pass a soft lubricated catheter through the nose and draw it forwards from behind the palate into the mouth. Tie string to a gauze swab and leave both ends long. Fasten one end to the tip of the catheter (Fig. 14A). After soaking the swab in one of the solutions mentioned withdraw the catheter through the nose so that the swab is pulled into the naso-pharynx (Fig. 14B). If there is difficulty a gloved finger behind the palate will enable the pack to be wedged in proper position. The ends of string are tied and fixed with strapping to the face. Very rarely bilateral choanal plugging and in addition packing the nose with gauze from in front is needed. Packs should be removed gently after 24 hours lest they cause otitis media.

Coagulant remedies. It is wise in difficult cases to use also remedies which may increase the coagulability of the blood (*page 126*).

Should bleeding recur in spite of all these measures ligation of the internal maxillary artery through the maxillary sinus and perhaps the anterior ethmoidal artery at the inner canthus will be needed.

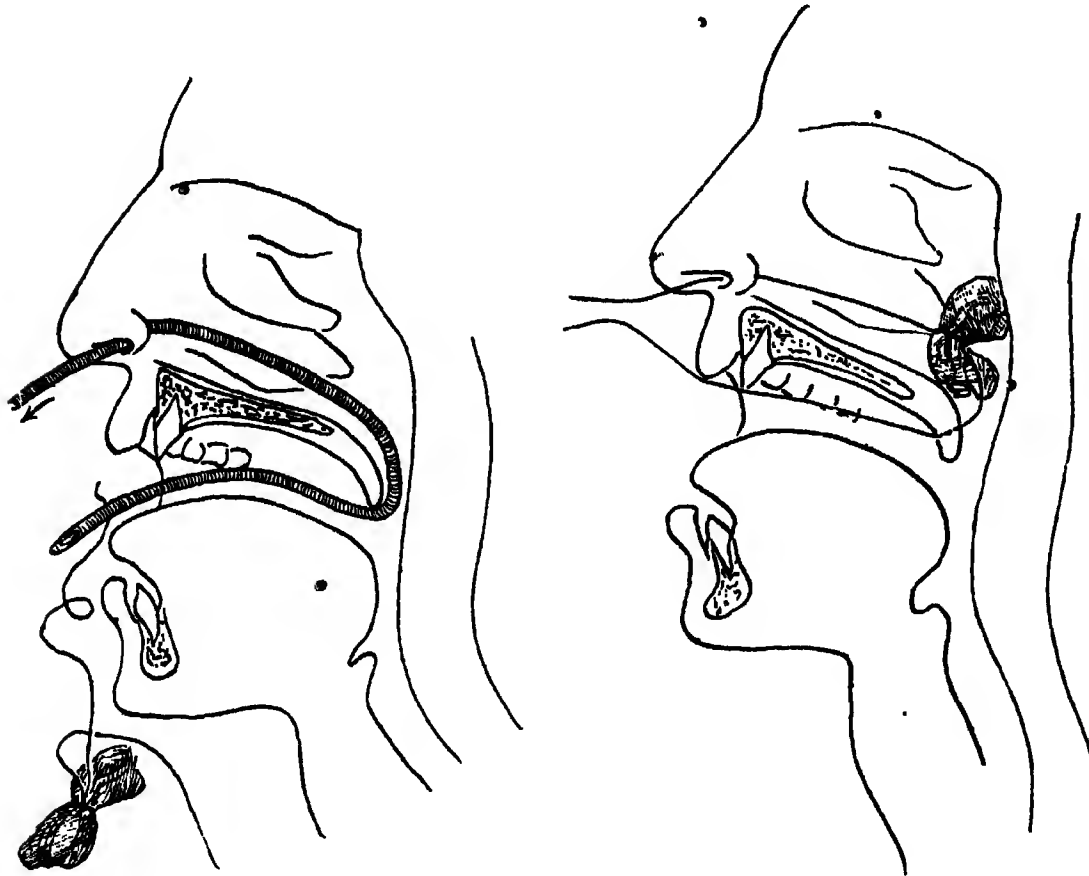


FIG. 14

A
Post nasal packing showing
catheter inserted and tied to
the pack.

B
Post nasal packing showing
pack pulled into position.

Epistaxis complicating hæmorrhagic disorders.—A less mechanical approach is advised when a bleeding disease is known to exist. The cautery and post-choanal pack should be avoided. When the bleeding point can be seen apply a piece of Calgitex gauze soaked in 1 in 1,000 adrenaline and hold it in place for five minutes. Let it come away of its own accord later. When no bleeding point can be seen or when there are many as in purpura a spray of thrombin as used in hæmatemesis (*page 88*) may be tried. Adrenaline may be added if its acidity, which would destroy thrombin, is buffered by sodium bicarbonate. Ten rag. (a small pinch) should be added to 10 ml. of Solution of Adrenaline Hydrochloride B.P. (*i.e.*, approximately 0.1 per cent.) and the resulting solution used to

dissolve the contents of a 10 ml. ampoule containing 5,000 units of thrombin. Blood transfusion may be needed.

HÆMOPTYSIS

The spitting of blood-tinged sputum or "streaking" usually calls only for reassurance as an emergency measure. In serious hæmorrhage from the lower respiratory tract, blood wells up into the mouth without much preliminary coughing. This is true hæmoptysis—the result of broncho-pulmonary hæmorrhage.

Sometimes the patient is uncertain whether the blood was coughed or vomited, and indeed some expectorated blood may be swallowed, as during sleep, and vomited later. Vomited blood is usually dark and may be acid (use *glazed* litmus paper to test it), whereas coughed blood is bright, frothy, mixed with sputum, and not acid.

It has been said that a patient never dies of hæmoptysis but after it, from the spread of tuberculosis caused by retention of infected blood clot. Though this is not strictly true even of tuberculosis, it emphasises the danger of failing to cough up the blood, for though death is rare from exsanguination it may occur from drowning. When hæmoptysis is profuse it is rapidly fatal as from a ruptured aneurysm, or is but a small part of the clinical picture of some other condition.

Treatment.—Only enough morphine should be given to allay anxiety, say, 11 mg. (gr. $\frac{1}{6}$), but the cough reflex must not be abolished. Since in the rare cases of fatal hæmoptysis death occurs within a few minutes, it is well to reassure the patient who has survived this period that he is not about to die. This is an important part of the treatment. An ice bag may be applied to the chest for its psychological effect.

The history, position of moist sounds and a unilateral sensation of warmth which the patient may notice will often indicate the probable side of bleeding. The patient should lie on this side to lessen the chance of blood entering the bronchi of the opposite side. Diaphragmatic movement will be increased on the lower side but this is relatively unimportant. If the patient prefers to lie flat then sandbags may be used to lessen movement.

There are no really effective remedies which can be given by mouth or injection to stop hæmoptysis. Most of the older coagulant substances such as congo red are ineffective. Coagulen—Ciba (a

thromboplastin preparation) 5 ml. intramuscularly may be tried but a transfusion of fresh blood is likely to be of more value.

If the patient is known to have a unilateral cavity or if a radiograph demonstrates this, a large pneumothorax (up to 1,000 ml.) may be induced. This has been done successfully, but is hazardous and may be ineffective because of adhesions. A simpler procedure is to induce a pneumo-peritoneum (*page 532*). Since the resulting rise of the diaphragm is bilateral, the affected side need not be known. If it is known, collapse can be increased by a phrenic nerve crush on the side of the cavity.

Bronchoscopy should not be done during hæmoptysis except by a thoracic surgeon who is ready to proceed further should more serious bleeding be activated.

ASTHMA

When called suddenly to a previously unknown patient in a paroxysm of supposed asthma, it is well to remember Chevalier Jackson's famous aphorism—"All is not asthma that wheezes."

"Asthma" with unilateral signs suggests bronchial obstruction. The sudden appearance of asthma in a middle-aged hypertensive subject suggests left ventricular failure ("cardiac asthma") (*page 162*). In such a patient, moist sounds in the lungs are a marked feature in contrast to the dry sibilant rhonchi of true asthma.

When real doubt arises as to the pulmonary or cardiac origin of the dyspnoea, an estimation of the circulation time may help. 2·4 G. of saccharine dissolved in 4 ml. of distilled water are injected into a cubital vein. The time taken until the patient tastes saccharine is noted (arm to tongue time). It is normal (up to 17 seconds) in asthma, but prolonged in left ventricular failure because of its slower passage through the congested pulmonary circulation.

Treatment.—One or more of the following should be used.

Adrenaline.—Injection of Adrenaline B.P. should be given subcutaneously at once. The earlier it is given, the more likely it is to be effective. Up to 0·3 ml. (5 minims) may be enough, but if wheezing persists this dose may be repeated every few minutes. It is important to make sure, by withdrawing the piston, that the needle point is not in a vein. Intravenous injections of undiluted

adrenaline (and inhalations of too strong solutions) cause pallor, anxiety, tremor and tachycardia (*page 44*).

In "status asthmaticus" a large dose, up to 7 ml. (120 minims) may be needed. The needle should be left *in situ* strapped with its syringe to the skin, and 0.06 ml. (1 minim) injected every 30 to 60 seconds. Alternatively, one of the slowly acting preparations may be used to keep up the effect (*see page 652*).

In intractable cases of "status asthmaticus" even 1 ml. of adrenaline straight off gives no relief and does not even cause a tremor. In these cases adrenaline can be given intravenously. A dose of 0.2 ml. Injection of Adrenaline B.P. should be drawn up and diluted with water to 1 ml. and this should be injected intravenously at a rate of not more than 0.1 ml. every half minute until relief is obtained.

Adrenaline Spray B.P.C. and N.F. or Adrenaline and Atropine Compound Spray B.P.C. and N.F. may be given as an aerosol (*for apparatus see page 604*). Inhalations of adrenaline solutions stronger than 1 in 1,000 should be used with caution, and only a few puffs of vapour inhaled.

Adrenaline-like substances. (*page 652*)

These are mainly isoprenaline (isopropyl-nor-adrenaline) and are issued as tablets for sublingual use (usually 20 mg.) and as a 1 per cent. spray solution. In an adrenaline-fast patient inhalation of up to 1 ml. of Isoprenaline Sulphate Compound Spray B.P.C. and N.F. is often very effective. Sublingual administration of a 20 mg. tablet of neo-epinine may abort an attack but is of little use in status asthmaticus and over-dosage causes unpleasant side effects.

Ephedrine.—Unless the patient objects to its side effects, 15 to 30 mg. ($\frac{1}{4}$ to $\frac{1}{2}$ grain) should be given night and morning.

Aminophylline.—When a patient fails to respond satisfactorily to adrenaline (and the patient in status asthmaticus is often refractory to it) a useful remedy is aminophylline (*see page 598*). It can be given as an intravenous drip using 1.5 G. in 1 litre of 5 per cent. dextrose. It is thought to sensitise the patient to adrenaline. The dose may be repeated in four to six hours.

Paraldehyde.—This may be given in doses of 3 to 4 ml. for an adult, preferably intramuscularly (*see page 597*). It is the best sedative for the asthmatic, for the others all tend to cause dangerous respiratory depression.

Morphine.—It is tempting to use morphine but this will nearly always be regretted, particularly in the elderly and in those who cough up plugs of mucus. Morphine depresses the respiratory centre and weakens the accessory muscles of respiration on which the patient is depending. It can certainly precipitate death in status asthmaticus. In a prolonged attack which is wearing the patient out, it is much wiser to use sodium phenobarbitone 0·2 G. (gr. 3) intramuscularly or paraldehyde as indicated above. Should these prove ineffective Dilaudid 2 mg. (gr. $\frac{1}{32}$) is preferable to morphine.

Chlorpromazine (Largactil).—Because of its depressant action on the cerebral cortex without respiratory depression this is often helpful in bringing an attack to an end. 12·5 to 25 mg. should be injected intramuscularly.

Pituitary extract.—Occasionally marked tachycardia will preclude the use of adrenaline and similar substances. In such cases, Injection of Pituitary (posterior lobe) B.P. 1948 (Pituitrin, Parke Davis) 3 to 5 units, or Injection of Vasopressin B.P. 1953. 5 to 15 units may be effective, but it is dangerous in elderly subjects who may have coronary ischæmia.

Posterior pituitary extract may be combined with adrenaline (*e.g.*, Kadamysin 1 ml. [formerly called Asthmolysin], Charles Zimmermann & Co., Ltd.).

A.C.T.H. and Cortisone.—A.C.T.H. should preferably be given by intravenous drip containing 20⁰ units and Aminophylline 0·5 G. (20 ml.) in 1,000 ml. of 5 per cent. dextrose. Up to 3 litres may be used in 24 hours. Treatment after this should be A.C.T.H. gel 30 units intramuscularly twice a day or Cortisone 50 mg. every four hours by mouth.

Other remedies.—It is wise to forbid flowers and smoking in the room and to give the patient a rubber pillow and mattress. An electric fan placed before the patient will help to make him more comfortable, especially if the atmosphere is warm and humid. Sometimes a prolonged attack which does not respond to the above measures will be relieved by the aspiration of mucus through a bronchoscope. Inhaling a mixture of oxygen 20 per cent. in helium at a pressure of 4 ml. of water, brings prompt relief, but this treatment is not readily available in Great Britain. Occasionally the treatment of asthma will be part of the

treatment of aspirin poisoning (*page 9*). Aspirin should generally be avoided by chronic asthmatics as it may make some of them worse. Individual cases may test the physician's ingenuity to the utmost. It must not be forgotten that some other condition such as pneumonia may underlie and be masked by "status asthmaticus." I have known attacks to be complicated by recurrent collapse of the lung, resulting from bronchial obstruction by thick mucus.

PLEURAL PAIN

Pleural pain, whether due to pleural tension or pleural friction, has the same characteristics. It is sharp and knife-like, worse on inspiration or coughing, most marked over the bases of the lungs and relieved by pressure over its site.

It may be associated with the onset of inflammatory or embolic changes in the lung, with pneumothorax and, very occasionally, with fracture of a rib from coughing. The treatment of the symptom is:—

1. To allay pain by morphine and cough by codeine or other drugs (*see page 146*). Spraying the skin over the painful area with ethyl chloride until blanching occurs will sometimes bring relief.
2. To immobilise the chest by strapping (which must go beyond the middle line front and back). This is not well tolerated by older patients with rigid chests. For them, a poultice is preferable.

INSTRUCTIONS FOR STRAPPING THE CHEST FOR PLEURAL PAIN.—Eight pieces of strapping, two inches wide, and six inches longer than half the chest girth are required. Sit the patient up and face his good side. Apply the centre of a piece of strapping to the mid-axillary region of the painful side. Hold one end of the strapping in each hand. Place the knee against the good side of the chest (or have an assistant press against this side). Ask the patient to hold his breath in full expiration, and while he does so, attach the plaster to the skin round the chest. Repeat the procedure until the eight strips are used, overlapping them a little.

A small artificial pneumothorax (250 ml.) may relieve the pain if friction be its cause, but is only advisable if pain is persistent and other measures have failed.

MASSIVE COLLAPSE OF THE LUNG

This is a form of absorption collapse which comes on comparatively suddenly, mainly as a result of an obstruction of a large bronchus by viscid secretion. It is usually a post-operative emergency and not always very sharply marked off clinically from, what is probably a rarer condition, true post-operative inhalation pneumonia. It has been shown to occur most often, after upper abdominal operations and is more frequent after local, rather than general, anaesthesia because of the heavier premedication when local anaesthesia is used. Morphine, by depressing the cough reflex, and atropine, by rendering bronchial secretion scanty and viscid, may play a part.

About 24 hours after operation the patient suddenly becomes dyspnoeic and cyanosed, and has discomfort in the chest which rarely amounts to pain. The degree of respiratory distress is very variable. The temperature rises to 101°F. There may be some sputum but it is never blood stained and the cough is often ineffective because of the pain it causes in the wound.

The most important single means of diagnosis is a radiograph of the chest, and this should be made in any patient who, soon after operation, shows evidence of respiratory trouble. It shows a uniform ground glass opacity extending outwards from the hilum and often clearing towards the periphery. When a large area of lung is involved, the heart moves over to the affected side and the diaphragm is raised.

In the absence of a radiograph, reliance must be placed on physical signs:—restricted movement and impaired resonance and air entry, together, when collapse is massive and unilateral, with evidence of mediastinal displacement to the affected side. Moist sounds are rarely present.

Treatment.—The object of treatment is to remove the bronchial obstruction and prevent retention of sputum. This may be done by bronchoscopy or even by aspiration through a soft rubber catheter passed via the nose after anaesthetisation of the larynx by a 2 per cent. amethocaine or Butyn (butacaine sulphate) spray. As this may not be readily achieved an equally good and often more rapid result can be obtained by postural percussion drainage.

One ml. of Isoprenaline Sulphate Compound Spray B.P.C. and N.F. should first be inhaled from an atomiser (*page 604*). The patient then lies on his back and is gently rolled back and forth

about a dozen times, the affected side being uppermost. A dose of Ammonium Chloride Mixture B.P.C. and N.F. or similar preparation is given, and the obstructing material may then be coughed up. If this fails violent coughing can be induced by Injection of Nikethamide B.P. 4 ml. intravenously. It is best preceded by a few



FIG. 15

Postural percussion drainage for massive collapse of the lung.

ml. of 2.5 per cent. thiopentone to make the patient drowsy. An assistant should support the wound with his hand. The manoeuvre may be repeated four-hourly and helped by springing of the ribs and heavy percussion of the affected side (Fig. 15). This may seem a formidable procedure for a patient with an upper abdominal wound, but the condition is a serious emergency and warrants vigorous treatment. Lest incomplete aeration should lead to pneumonia, it is wise to give penicillin.

Oxygen may be given, but CO_2 is contra-indicated as the hyper-ventilation it causes may lead to aspiration of material into the smaller bronchi. In desperate cases with great pain and dyspnoea, and marked mediastinal shift due to high negative intrapleural pressure, an artificial pneumothorax sufficient to

restore the intrapleural pressure to normal will give marked relief.

PULMONARY EMBOLISM

Pulmonary emboli are of two kinds:—(a) small and often multiple; (b) large and single. Thrombotic pulmonary emboli originate in the peripheral veins when they are the seat of thrombosis (though rarely from phlebitis in varicose veins) or from post-operative thrombi in the pelvic and abdominal veins. Such emboli are more likely to reach the left lung than the right because the left pulmonary artery is more in line with the main pulmonary artery than is the right. Emboli may also come from the right side of the heart in bacterial endocarditis and from mural thrombi in the right auricle in auricular fibrillation or in the right ventricle following a cardiac infarct.

Thrombosis follows embolism, and a pulmonary infarct develops after a short interval. In some cases of congestive cardiac failure, pulmonary thrombosis without previous embolism is responsible for the infarct. The symptoms produced by a small embolus are pleural pain followed by hæmoptysis. In less sudden cases, rise of temperature and pulse rate may be observed first. Sometimes there is only tachycardia and a sense of oppression in the chest. Physical examination reveals either nothing at all or perhaps a friction rub. X-ray examination may show an infarct, rarely as a classical triangular shadow and more often as basal clouding with elevation of the diaphragm. With small emboli X-ray evidence may be absent. Small pulmonary infarcts are, in fact, often atypical. They more often complicate medical than surgical illnesses and as they do not cause urgent symptoms they may be overlooked, particularly if there is also congestive cardiac failure. If the bronchial circulation is good a small embolus may not cause an infarct. It is more likely to do so when the general circulation (including the bronchial circulation) is embarrassed.

Massive pulmonary embolism produces a cardio-vascular emergency of the first order (*see also pages 163 and 462*). The patient is usually elderly and has thrombosis of the calf and leg veins from recumbency during a medical or surgical illness. He is suddenly seized with a feeling of suffocation and has substernal oppression, but no severe pain and no radiation of pain as in coronary occlusion. Symptoms quickly become maximal and the patient is shocked and cyanosed and loses consciousness.

Sometimes there is great restlessness as he throws himself about in his fight for breath. A typical symptom is an urgent desire to defæcate. Only when 60 per cent. of the pulmonary artery lumen is occluded does the cardiac output fall, and so a large elongated thrombus in the pulmonary artery may be symptomless until it suddenly twists on itself and occludes the lumen. Death is fairly sudden, though never so quick as in thrombosis of a main coronary artery. Many post-operative cases have occurred at the tenth day when the platelet rise is at its height and the patient begins to move more freely in bed.

On examination the heart sounds are feeble. Sometimes there is auricular fibrillation and a loud pulmonary systolic murmur. Air-entry is normal at all areas and there are no râles. From this picture, a confident clinical diagnosis can be made, but in doubtful cases the E.C.G., if taken early, is helpful, though lesser degrees of embolism may fail to show any changes. Præcordial leads may be diagnostic since they show inversion of T_u waves over the right ventricular region, *i.e.*, in leads V 1, 2 and 3. In the standard leads there is a prominent S wave in lead 1 and a prominent Q and inversion of T in lead 3. T₂ is flattened. These changes are important in distinguishing the condition from posterior cardiac infarction in which the S wave in lead 1 is not prominent but in which Q is prominent in lead 2 and the R.T. segment elevated in lead 3.

When pulmonary embolism is not rapidly fatal, the condition known as acute "cor pulmonale" may develop. It arises from dilatation of the right heart following increased pressure in the pulmonary circuit. It is characterised by prominence and pulsation of the second and third left intercostal spaces just to the left of the sternum, *i.e.*, in the area over the dilated pulmonary artery. There may be a systolic murmur and a loud pulmonary second sound and often a friction rub and triple rhythm. The jugular veins are dilated and pulsating. This state develops suddenly with dyspnœa and a feeling of oppression in the chest in a patient who has already shown signs of small pulmonary emboli. If shock is marked it may obscure the picture completely. The condition may subside in a few hours, or last a few days until death occurs.

Diagnosis.—The other acute "chest calamity" with which pulmonary embolism may be confused is coronary thrombosis. A history of anginal attacks, a marked fall of blood pressure,

and the absence of phlebo-thrombosis are points in favour of coronary thrombosis. Marked cyanosis, dyspnœa and restlessness are in favour of pulmonary embolism, especially when there is a possible source of an embolus. The pain of pulmonary embolism rarely spreads to the arms as does that of angina. Blood-stained sputum suggests embolism. The E.C.G. evidence may be helpful (*see page 134*). Sudden death following a recent injury makes clear differentiation of these cases of medico-legal importance since embolism might be obviously attributable to the injury whereas coronary thrombosis would not.

Treatment.—Small pulmonary emboli are warnings of a more severe catastrophe. Thrombosis of leg veins (*see page 463*) from which such emboli mostly originate is a fairly silent process and so should be looked for carefully and local signs such as that of Homan sought (calf pain on dorsi-flexing the foot with the knee straight). If found the physician should not neglect the help of his surgical colleagues. Ligature of the femoral vein or partial occlusion by a clip (*see page 463*) or even ligature of the inferior vena cava may be needed. Anti-coagulant therapy (*page 595*) should be used until the patient is up and about with a view to preventing further clot formation.

When massive pulmonary embolism occurs, oxygen and nikethamide should be given. It has been suggested that part of the clinical picture results from reflex coronary spasm and for this reason intravenous injection of papaverine 65 to 130 mg. (gr. 1 to 2) with atropine 1 mg. (gr. $\frac{1}{64}$) is recommended.

If the patient survives the initial shock heparin should be given (*see page 595*) in the hope that, by preventing addition to the embolus by thrombosis, sufficient circulation will be maintained to save life.

FAT EMBOLISM

This is considered here because many of the manifestations are pulmonary. It usually complicates a bone injury in a young adult and is probably commoner than is generally supposed being mistaken for concussion, shock and traumatic uræmia. In a typical case a patient who has sustained a fracture is admitted to hospital after a rough journey with the limb improperly immobilised. Following manipulation under anæsthesia he remains unconscious or lapses into coma. In milder cases periods of delirium and stupor alternate

with full consciousness. This alternation is caused by crops of cerebral emboli which act, not mechanically, but by liberation of fatty acids which destroy the walls of small vessels. The origin of fat globules may be the bone marrow and body fat but it is thought that they may also arise from conglomeration of plasma lipoids.

Dyspnœa and cyanosis are usually present. Fine râles can be heard, and an X-ray film may show diffuse hazy shadows (venous or pulmonary fat embolism). Pyrexia is usual and, in the absence of pneumonia, should raise one's suspicions. Fits may occur but focal nervous signs are unusual. If they are present the diagnosis from intra-cranial hæmatoma may be difficult, since in both conditions there may be a "lucid interval." In a doubtful case intra-cranial exploration would be wise.

(Chest symptoms similar to those described have followed salpingography by iodised oil, either immediately or several days later. In such cases, X-ray films of the chest have shown dense linear basal opacities caused by opaque oil in the smaller pulmonary arteries. Sodium acetrizate, 30 per cent. (Diaginol) carries no such risk).

Confirmatory evidence should be looked for in the skin, fundi and urine (arterial fat embolism). Petechiæ occur at the base of the neck, in the axillæ and in the conjunctiva of the lower lid. The fundi show perivascular hæmorrhages and white patches of œdema. The appearance of fat globules in the urine is a late and inconclusive sign. Since fat floats in urine, only the last drops voided may show it. If these signs are absent in an otherwise clinically suggestive case then embolisation by fragments of bone marrow rather than fat globules may be responsible. An emergency call may be occasioned by a sudden worsening of the patient's condition and the development of cerebral symptoms.

Treatment.—This is symptomatic by the administration of oxygen and analeptics (*page* 608). Immobilisation of the fracture should be complete. Although recent work suggests that fat globules are not the only factor causing symptoms it would seem reasonable to try to disperse them by giving 10 ml. of 20 per cent. sodium dehydrocholate (Dehydrocholin B.D.H.; Suprachol. Richter) by slow intravenous drip every two hours. Heparin (50 mg. intramuscularly) has been suggested because it will clear post-prandial lipæmia. It might be tried if not otherwise contraindicated.

SPONTANEOUS PNEUMOTHORAX

When this causes urgent symptoms it is usually large and complete. Exertion may or may not determine its onset and the patient is often a young healthy person. There is sudden sharp pleural pain, followed, as the lung collapses, by dyspnoea.

The classical signs are lack of movement on the affected side with resonance and auscultatory silence, together with displacement of the apex beat to the opposite side. Sometimes distant breath sounds with a peculiar metallic or amphoric quality are heard and are diagnostic. If the pneumothorax is small, pleural pain alone may constitute the emergency and accurate diagnosis will depend on X-ray examination. The urgency of symptoms arising from a pneumothorax is determined by:—

1. The tension within it.
2. The degree of mediastinal displacement.
3. The presence of bleeding (*Hæmo-pneumothorax*, page 139).
4. The severity of the pleural pain.

Although symptoms are most marked when intra-pleural pressure is high, they may be urgent also when the pressure is negative if the patient's vital capacity is diminished by emphysema.

TENSION PNEUMOTHORAX.—This “*pneumothorax suffocans*” is the most serious type. It renders one side of the chest immobile and bulging, and constitutes a real threat to life because, by pressure on the great veins, it interferes with the return of blood to the heart. It is shown by:—

1. Displacement of the apex beat to the opposite side.
2. Increasing cyanosis and dyspnoea.
3. Increasing rapidity of the pulse.
4. Excessively high positive pressure.

Downward displacement of the liver by a right-sided tension pneumothorax is a late sign as is also the change in the percussion note from resonant to dull when the tension is great. Such evidence should not be awaited.

The mechanism of tension pneumothorax is disputed. Pneumothorax following needling of the chest is usually simple but very rarely a tension pneumothorax may develop. In such a case it seems possible that the hole in the lung may open with inspiratory expansion and close on expiration much in the same way that a tyre puncture becomes more obvious on inflation.

Another mechanism which applies particularly to tension pneumothorax following injury is that an alveolus ruptures and causes interstitial emphysema of the lung and later of the mediastinum. The mediastinal air subsequently ruptures into the pleural space. Tension in the pneumothorax is built up during the forced expiration of coughing. This mechanism will explain pneumothorax which appears on the side of the chest opposite

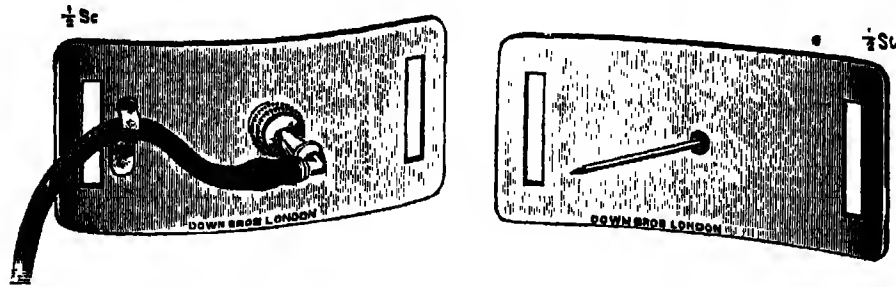


FIG. 16

Foster-Carter's needle.

to the injury. The presence of subcutaneous emphysema of the neck in these cases supports this theory.

A third and most likely mechanism is the same as that causing a tension cavity, namely, a valve-like action of the bronchus. When the bronchus leading to the rupture on the lung surface is kinked during expiration, or partially obstructed by caseous bronchitis or bronchial spasm, it may allow air to pass on inspiration but not on expiration, and so enable a positive pressure in the pleural space to be built up.

Treatment.—Morphine, brandy and oxygen are the first things to use. If respiratory distress persists, a needle should be inserted in the fifth or sixth space in the mid-axillary line and connected with a manometer. If the pressure is positive, air should be aspirated by a reversed pneumothorax apparatus. In extreme urgency, a needle by itself will release air and save the patient's life. Should urgent symptoms recur, it is best to leave the needle in the chest and connect it to a rubber tube which dips under water. This should be well below the level of the needle to obviate any risk of the water being aspirated into the chest. The pressure at which the air blows off can be adjusted by altering the depth to which the tube is immersed. In this way air can be expelled via the needle, but cannot enter it. This is insufficient in some cases and continuous suction may be needed (page 533). The needle may be kept in position by

inserting it through a piece of cork which is strapped to the chest. Foster-Carter's needle (Fig. 16) is an improvement on this. It is attached to the chest wall by a metal or polythene plate and the length within the chest can be adjusted by a screw clamp. In the absence of a filter pump (*page 533*), gentle suction sufficient to relieve the patient's distress can be maintained by a simple apparatus devised by Mariott and Foster-Carter (*page 534*).

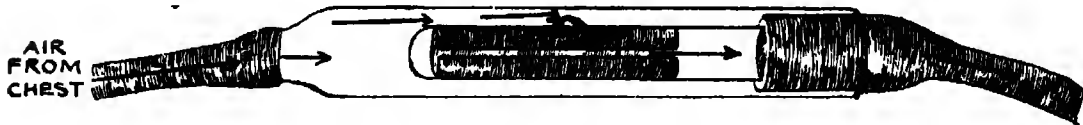


FIG. 17

Reversed non-return valve from Army blood transfusion set.

If a patient with a tension pneumothorax has to be moved, as after an accident, the non-return valve from an Army blood transfusion set is a useful device. This normally lets air into the blood bottle and prevents its escape, so it must be attached to the needle in the chest in the reversed position (Fig. 17).

Even when air has ceased to escape, it is not safe to leave the patient, for symptoms may recur, and fluid, particularly blood, may accumulate.

SPONTANEOUS HÆMO-PNEUMOTHORAX

In every case of spontaneous pneumothorax, the physician should bear in mind the possibility that blood as well as air may be escaping into the pleural cavity. The blood comes from the parietal end of the adhesion torn by the collapsing lung. It is more likely to stop if it is low pressure bleeding from the lung than if it comes from an artery on the chest wall.

After the sudden onset of pneumothorax there is a period of temporary improvement lasting some hours or even days, and then increasing pallor, increasing rapidity of pulse, dyspnoea and restlessness, indicating internal hæmorrhage. Physical signs of air and fluid in the pleural cavity will be found. Abdominal symptoms resembling a surgical emergency are more apt to occur in hæmo-pneumothorax than in pneumothorax, because of irritation of the diaphragmatic pleura by blood. It is wise, therefore, to put a patient with a spontaneous pneumothorax on a half-hourly pulse chart.

A surprising amount of blood may be found in the pleural cavity even when the bleeding point is small. This is because of the suction of the negative intra-pleural pressure and the fact that movement prevents clotting by defibrinating the blood.

Treatment.—The patient should be treated as for spontaneous pneumothorax and transfused if necessary. Aspiration of blood preferably through a large thoracoscopy cannula should not be delayed longer than a few hours lest fibrin deposit interfere with expansion of the lung later.

When it is clear that bleeding continues or if there has been delay so that clotting is likely it is best to call a thoracic surgeon.

MEDIASTINAL EMPHYSEMA (HAMMAN'S SYNDROME)

This results from rupture of pulmonary alveoli giving rise firstly to interstitial emphysema of the lung, and later, as the air spreads, to mediastinal emphysema. Numerous conditions may cause this rupture, such as trauma, including artificial pneumothorax therapy (*page 25*) and blast injuries, and all the causes of increased intrapulmonary pressure, such as cough, and straining with the glottis closed. Sometimes an alveolus appears to have ruptured spontaneously in the absence of any known cause of increased pressure. Rarely air has reached the mediastinum from a perforated duodenal ulcer.

If of sufficient amount, mediastinal air may escape:—

1. Into the neck (Subcutaneous emphysema).
2. Into the retro-peritoneal tissues.
3. Into the pleural cavities (Spontaneous pneumothorax).

Diagnosis.—Small amounts of air in the mediastinum are symptomless. Large amounts cause pain and retrosternal oppression like that of coronary disease, and also dysphagia. Diagnosis may be difficult unless subcutaneous emphysema occurs. This, by lowering the tension, alleviates the pain. Other important signs are obliteration of the cardiac dullness, and peculiar crackling and bubbling sounds synchronous with the heart beat and accentuated in systole ("pericardial knock"). Occasionally the patient is himself aware of the peculiar sounds. Pericarditis and pneumo-pericardium are mimicked. The intensity of the sounds bear no relationship to the severity of the symptoms. X-ray examination is not of much value in demonstrating mediastinal air

unless this is large in amount. An E.C.G. may be helpful in excluding coronary occlusion.

More urgent symptoms are caused by pressure on the big veins so that return of blood to the heart is impeded. Dyspnoea, cyanosis and pulmonary oedema result, and death may occur from circulatory standstill. Convulsions from cerebral congestion are possible. These symptoms are the result of "air block" in which there are two elements, obstruction to the return of venous blood to the heart and interference with respiratory movements of the lung. I have seen the escaped subcutaneous air constitute an emergency because the patient's face and neck became rapidly distended, causing respiratory obstruction.

Treatment.—This becomes urgent when pressure symptoms appear. Probably the most effective measure is to incise the tissues at the root of the neck, introduce a catheter, and apply suction. Tracheotomy instruments should be prepared. If unilateral lung disease is present, it is reasonable to assume that the air leak is on the diseased side, and to try the effect of collapsing this lung by artificial pneumothorax. On the only occasion in which I had to deal with this emergency, this method was successful.

ACUTE RESPIRATORY INFECTIONS IN EMPHYSEMA

When a bronchitic and emphysematous patient develops a chest infection pyrexia and anoxia may quickly make him cyanosed, drowsy and disorientated. Arterial blood analysis will show that he has CO_2 retention, acidæmia and anoxæmia—the characteristic triad of respiratory failure. He may die suddenly and unexpectedly. The following are the important aspects in treatment.

(1) OXYGEN.—The respiratory centre of the emphysematous patient becomes relatively insensitive to an increase of CO_2 in the inspired air and breathing is governed more by the effect of anoxia on the carotid and aortic chemoreceptors. When oxygen therapy during an acute respiratory infection removes the stimulus of anoxia the tension of CO_2 in the arterial blood (pCO_2) rises. If this rise is rapid and an uncompensated respiratory acidosis occurs then CO_2 narcosis (mental symptoms, lethargy and coma) may appear. Anoxia, however, is even more dangerous. It too can cause mental clouding and also peripheral circulatory failure. In

practice we have to steer our patient between the Scylla of anoxia and the Charybdis of CO₂ narcosis. This is achieved by giving oxygen *gradually*. Too rapid oxygenation causes a rapid rise of pCO₂ and the resulting fall of blood pH is too rapid for the kidneys to compensate immediately.

Oxygen is best given to these patients by nasal tubes at 1 to 3 litres a minute. An oxygen tent is inadvisable as it cannot easily achieve gradual oxygenation because at low rates of flow the temperature rises unduly. Also a tent is hazardous should the oxygen supply fail. As anoxia is more serious than CO₂ narcosis oxygen should be persisted with even though it causes some mental confusion. If coma supervenes when oxygen is given the rate of flow should be cut down. The patient can be roused from the coma of CO₂ narcosis by the rapid intravenous administration of 4 ml. of Injection of Nikethamide B.P.

(2) ANTIBIOTICS.—Until sputum tests indicate otherwise it is best to give Streptomycin 1 G. daily with a view to eliminating *H. influenzae* and Penicillin 1 mega unit daily.

(3) SEDATIVES.—Paraldehyde (*page* 597) is best since it does not cause respiratory depression. Morphine and barbiturates should be avoided since they may abolish all sensitivity of the nervous control of breathing. Should morphine have been given already (and a specific question should be asked about this) its effects can be counteracted by nalorphine (*page* 14).

(4) RELIEF OF OBSTRUCTION TO BRONCHIOLES.—This is aided by Ephedrine 16 to 65 mg. (gr. $\frac{1}{4}$ to 1) three times a day to relieve bronchospasm and by coughing. The drowsy patient may fail to cough up obstructing plugs of mucus and pus. Inhalations of an aerosol of Alevaire or Trypure (*page* 123) may loosen the sputum but they should be avoided if the patient is too weak to cough it up. Coughing can be initiated by the intravenous administration of Injection of Nikethamide B.P. 2 ml. but it is not necessary if the nurse can get the patient to clear his chest periodically.

PNEUMONIA AS A MEDICAL EMERGENCY

In these paragraphs, acute lobar and acute lobular or bronchopneumonia are not differentiated. All acute types of pneumonia are considered together and may be equally urgent, except virus pneumonia ("primary atypical pneumonia"). This is a less

serious disease and unlikely to present an emergency. (*For pneumonia in children, see page 294*).

In all patients over 40 years of age, the onset of pneumonia should be regarded as an emergency. Positive diagnosis is often difficult in the old and debilitated ("asthenic pneumonia"). Antibiotic treatment should be started on suspicion and time should not be lost awaiting obvious clinical signs. In younger patients the urgency depends on the severity of the attack and the previous state of the patient's essential organs.

Pneumonia is by nature a local disease of the lung which strives to become general and in so far as the evidence of bacteriæmia and involvement of other organs is greater, so the urgency of the condition increases. Pneumonia may cause an emergency situation:—

1. By presenting as a grave undiagnosed pyrexial illness.
2. Because of some urgent symptom arising in a patient known to have pneumonia.

Diagnosis.—This may be difficult in the early stages. In a typical case the onset is sudden with shivering and rigor, a high temperature, a quick bounding pulse, and a hot dry skin. Evidence pointing to trouble in the chest, such as rapid breathing, pleural pain, and a hard cough with viscid rusty sputum makes the diagnosis easier. Dulness on percussion and noisy breathing confirm this, but it must be remembered that signs may be atypical because the consolidation is deep or the bronchus blocked. In "central pneumonia" diminished movement on the affected side is an important sign.

Management.—There are certain urgent features in the general management of the case. If it seems doubtful whether a very ill patient can be nursed at home, removal to hospital should be arranged early while there is still hope, rather than later as a result of despair and defeat. Should home be decided upon, choose early the best room. This may be downstairs to save wear and tear on the legs of relatives (and the doctor). Pneumonia occurring at holiday times calls for foresight in laying in a supply of essential drugs and oxygen.

It is wise to be forearmed about prognosis by knowing the causal organism. Sputum must be saved early in the attack so that the organism may be identified and kept for sensitivity tests.

Treatment.—Penicillin, 1 mega unit in 24 hours, is the antibiotic of choice.

Oxygen should be given early. In the first few days "sleep at any price" is a justifiable maxim and morphine should not be withheld. For feeble patients pethidine is safer.

Urgent symptoms and complications.

Many urgent symptoms are discussed elsewhere: pleural pain (*page* 130), acute abdominal symptoms (*pages* 65 *and* 287), mediastinal emphysema (*page* 140) and acute mental symptoms (*page* 230). Pneumonia may be a complication of other emergencies, such as diabetic coma, barbiturate poisoning and cerebral vascular accidents. Jaundice is a rare but serious complication and should be treated as described under hepatic failure (*page* 82).

Abdominal distension or meteorism is serious because it interferes with the action of the diaphragm. The rectum should be examined, and if full of *fæces*, an enema given. A flatus tube should then be passed and retained while drugs are used. Carbachol—a parasympathetic stimulant—0.25 to 0.5 mg. is given by subcutaneous injection (it must not be given intravenously). If these fail, Injection of Pituitary (posterior lobe) 5 units with eserine salicylate 0.65 mg. (gr. $\frac{1}{100}$) or Prostigmin 0.5 mg. subcutaneously may be tried.

While the urgent nature of the illness may be obvious from delirium, severe pain and other symptoms, these may be absent even in a severe case. Because of pre-existing mental disorder a patient may show little reaction to pneumonia. Alternatively, a previously normal brain may be so poisoned that the patient fails to show distress and remains calm and comfortable. Euphoria in pneumonia may therefore be an ominous signal. As an old aphorism warns us—"Beware of pneumonia that smiles at you."

Overwhelming infection is shown by evidence of pericarditis and meningitis. The latter should be looked for, especially since, if penicillin is going to be used, it is ineffective unless given intrathecally.

Medical shock in pneumonia. (See also *page* 152)

This is usually described by the less arresting term "peripheral circulatory failure." It arises essentially from disproportion

between the capacity of the vascular bed and the volume of the blood in it.

In traumatic shock, diabetic coma and severe diarrhoea, the circulation may fail because the circulating blood volume falls. In the shock of acute infections, capillary dilatation from paralysis due to bacterial toxins is thought to be the mechanism.

The state of the peripheral circulation may be gauged clinically by observing the pulse, the arterial blood pressure, the venous blood pressure (as shown by the height above the heart at which the hand veins collapse), and by looking for capillary pulsation (as shown by gentle pressure on the finger nail or by noting rhythmic dimming of the illuminated finger pulp).

In medical shock the pulse is rapid and thready, the arterial and venous pressures are low, capillary pulsation may be present, the breathing is shallow, and the skin cold and moist. This picture complicating pneumonia was formerly ascribed to cardiac failure. Although toxæmia and anoxæmia may damage the heart muscle, it is peripheral and not central circulatory failure which is the main factor. The venous return is inadequate to maintain the circulation. "The well runs dry."

Treatment.—Warmth is important. The blood supply to the brain may be improved by raising the foot of the bed or binding the limbs. Injection of Nikethamide 2 ml. intramuscularly every hour is useful for the associated respiratory depression. Injection of Adrenaline B.P. 0.3 ml. (m 5) every four hours or Injection of Pituitary (posterior lobe) 0.5 ml. every four hours may be tried but no blood pressure raising drugs are very effective in pneumonia.

Oxygen in high concentrations is very helpful. Little improvement can be expected until the infection and consequent toxæmia are brought under control. If this is not done, circulatory failure will progress to death.

Digitalis is valueless in pneumonia except when auricular fibrillation supervenes (*see also page 155*). Analeptics should not be used routinely, but when there is evidence of failure of the respiratory centre nikethamide and leptazol are helpful (*see page 609*).

COUGH AS AN URGENT SYMPTOM

Prolonged irrepressible coughing may be the reason for an emergency call. If purely symptomatic treatment is called for one of the following remedies may be tried.

- (1) Amidone in a linctus, *e.g.*, Physeptone Linctus (Burroughs Wellcome) 4 ml. (m 60) (=2 mg. Amidone) every three or four hours for an adult.
- (2) Pholcodine (morpholinylethylmorphine), *e.g.*, Ethnine (Allen and Hanburys) 8 ml. (m 120=8 mg. Pholcodine) every three or four hours for an adult.
- (3) Becantyl (Horlicks), a butyl naphthalene monosulphonate. Adult dose 8 ml. (m 120).
- (4) Diamorphine (Heroin) *if obtainable*.
Linctus Heroin B.P.C. (gr. $\frac{1}{20}$ in m 60 or 3 mg. in 3.5 ml.).
Dose 2 to 8 ml. (30 to 120 m).
Heroin hydrochloride 5.4 to 11 mg. (gr. $\frac{1}{12}$ to $\frac{1}{6}$) may be given by injection but should be avoided in younger patients lest the pleasant feelings it causes give rise to addiction.
- (5) Toclase, Pfizer (Carbetapentone).
Toclase hydrochloride (issued as a syrup containing 15 mg. in 10 ml.). Daily dose 60 to 90 m (40 to 60 ml.).
Toclase citrate (issued as 25 mg. tablets). Daily dose 75 to 100 mg. (citrate is less potent than hydrochloride).
- (6) Slow inhalation of 70 per cent. ethyl alcohol from a gauze mask is often a great help.
Paraldehyde (*see page 597*) will enhance the effect of these drugs without further depression of the respiratory centre.

COUGH SYNCOPE

Paroxysmal coughing may, by impeding the venous return and pulmonary circulation, so decrease cardiac output that syncope occurs from cerebral anoxæmia. Its mechanism is similar to that of Valsalva's manœuvre in which forced expiration against a closed glottis causes vertigo and syncope.

The subject is rarely a woman but generally a middle-aged emphysematous and plethoric (but not hypertensive) man who, after a fit of violent coughing, and in rare cases after only a few coughs, becomes unconscious for a few seconds. Those who have much sputum rarely have syncope on coughing. In minor attacks there is simply giddiness and Charcot, who first described the condition, called it "laryngeal vertigo." The emergency soon rights itself and between attacks no special treatment is needed though tobacco which causes the cough should be avoided. The patient should try to cough gently as violence in coughing seems

to be the important factor. When unconsciousness follows a single cough in a young adult it is thought that the condition is epilepsy with cough as the aura.

HICCUP

(For hiccup complicating anæsthesia see page 442).

In hiccup sudden involuntary contractions of the diaphragm occur repeatedly. The resulting inspirations are cut short abruptly as the vocal cords come together and the characteristic "hic" is caused. When hiccup is rapid and persistent it may exhaust the patient and call for urgent treatment. This should be directed to the cause be it abdominal (peritonitis), thoracic (pleurisy), cerebral (encephalitis) or toxic (uræmia). In really persistent hiccup there is often no organic basis and the cause is psychogenic.

Hippocrates said that a bout of sneezing might cure hiccup and so snuff may be given. If this and other homely remedies such as breath holding and drinking cold water have failed the following measures should be tried.

1. Pulling the tongue forwards.
2. Oil of cloves on a lump of sugar or a strong carminative such as:—

Menthol	0.5 G.
Aromatic Spirit of Ammonia						} 30 ml. of each
Spirit of Chloroform						
Tincture of Ginger						

4 ml. (one teaspoonful) in water taken as strongly as possible.
3. CO₂. Stimulation of the respiratory centre by CO₂ will sometimes end an attack. It should be administered early before the patient is exhausted and preferably by an anæsthetist. A simple method is to let the patient breathe in and out of a length of wide rubber tubing (as from an anæsthetic machine). This prolongs the dead space and so causes rebreathing of CO₂. The capacity of the tubing must be less than the tidal volume or else anoxia will result. (For a tidal volume of 500 ml. and tubing of 2 cm. diameter a length around 150 cm. would be needed).
4. Atropine 0.4 to 1.2 mg. (gr. $\frac{1}{150}$ to $\frac{1}{50}$) or Hyoscine 0.3 mg. (gr. $\frac{1}{200}$) intravenously.
5. Inhalation of amyl nitrite.

6. Pethidine 50 mg. intravenously and repeated in an hour if need be.
7. Chlorpromazine (Largactil) 25 to 50 mg. intravenously with the patient in bed.

When drugs have failed for several days the phrenic nerve itself may be attacked preferably in the left side because spasms are more often left sided, and cause more distress than when right sided.

Digital pressure may be used first but only to show which side is mostly affected. It will not cure the condition. Put a pillow under the neck to cause slight anterior arching. Place the first two fingers at the posterior border of the sterno-mastoid just above the clavicle and push inwards and backwards for not more than two minutes. Spasms may cease within a minute but will recur.

Surgical exposure should be made under general anæsthesia so that all roots of the phrenic nerve and the main trunk can be crushed. Branches communicating with the vagus and sympathetic should be divided. A ligature should be left round the nerve so that it can be easily found again if need be.

USELESS REMEDIES.—Amphetamine, quinidine, ethyl chloride spray, hypnosis and deep anæsthesia have all been tried and found useless.

C. ALLAN BIRCH.

CHAPTER VIII

Cardio-Vascular Emergencies

EMERGENCIES are common in cardio-vascular disease. They range through all grades of severity, from the annoyance of nocturnal extrasystoles sufficiently numerous to provoke alarm and prevent sleep to the most profound degrees of pain, shock and collapse. Death may threaten in a matter of seconds or minutes. The doctor in consequence may know little or nothing of his patient until faced with the actual emergency. It demands a quick eye and a ready judgment to assess the seriousness of the situation.

A simple classification of the common circulatory emergencies based on the predominant symptom is therefore useful. Admittedly, such a system has its imperfections and must be used with discretion as one symptom may dovetail with another. The pain of a coronary thrombosis, for instance, may be masked by the urgent dyspnoea of left ventricular failure. Conversely, an attack of paroxysmal tachycardia—a common cause of palpitation—may be sufficiently severe in the elderly to cause angina or even syncope. Palpitation may then be of less significance to the patient. These are exceptions. To be appropriate, immediate treatment must be based on underlying causes which unfortunately are sometimes difficult to determine in the urgency of the situation. As a general rule the leading symptom justifies such a classification as the following:—

- (1) Syncopal attacks:—(a) simple faint, (b) carotid sinus syncope, (c) postural hypotension, (d) Gowers' syndrome, (e) Stokes-Adams seizure.
- (2) Collapse and shock.
- (3) Palpitation:—(a) extrasystolic irregularity, (b) paroxysmal auricular tachycardia, (c) paroxysmal ventricular tachycardia, (d) paroxysmal auricular flutter, (e) paroxysmal auricular fibrillation.
- (4) Dyspnoea:—(a) acute left ventricular failure ("cardiac asthma"), (b) acute right ventricular failure (pulmonary embolism), (c) cardiac tamponade.

- (5) Præcordial pain:—effort angina and coronary thrombosis.
- (6) Peripheral embolism.

SYNCOPAL ATTACKS

Syncopal attacks arise as a result of acute cerebral anæmia. The essential feature is an abrupt decrease in the volume of blood in active circulation. A profuse hæmorrhage produces symptoms which differ only quantitatively from those of a simple faint. In hæmorrhage blood is lost to the exterior; in fainting it is temporarily pooled in the venous reservoirs of the splanchnic area.

(a) **A simple faint** is best treated by loosening the collar, and by placing the patient flat on his back. This facilitates the return of blood to the heart and tends automatically to correct the cerebral anæmia. An even better effect may be obtained by supporting each foot some inches from the ground, thereby allowing gravity to aid the return of blood to the heart. A more effective manœuvre is to place the patient in a modified Trendelenburg position with the knees and ankles above the level of the head. Consciousness usually returns rapidly. Massage from the periphery towards the heart has a similar effect. Warmth aids recovery. Hot tea or coffee are generally acceptable and are probably more serviceable than whisky or brandy.

Prevention depends on the detection of exciting causes. Emotional stress in certain excitable individuals should be avoided. Pain precipitates attacks in other people. Long hours without food, the erect posture, a hot, stuffy atmosphere and disagreeable sights or sounds are factors known to upset vaso-motor control and cause a simple faint.

(b) **Carotid sinus syncope** is a special variety of fainting attack attributable to a hypersensitive state of the carotid reflexes. That gentle pressure with the finger tips over one or other carotid sinus can reproduce constantly in a matter of seconds a train of symptoms terminating in a syncopal attack exactly similar to that to which the patient is liable, provides good evidence of the hypersensitive state of the sinus mechanism, and serves to distinguish it from other forms of syncope. Pronounced cardiac slowing with or without a fall in blood pressure is a common feature. Injection of Adrenaline (0.25 to 0.5 ml.) subcutaneously is a very effective remedy. A small tumour or gland in the neck may be a source of irritation responsible for the attacks, and require removal. Fortunately, hypersensitivity of the carotid sinus is not necessarily permanent, and measures designed to improve the patient's general health are often effective.

(c) **Postural hypotension** causes syncope very readily when the erect posture is assumed. Postural fainting is still apt to

occur even after injecting adrenaline which, though it increases the heart rate, does not restore vasomotor control in these patients. Ephedrine 50 mg. (gr. $\frac{3}{4}$) by mouth, even as often as every two or three hours, does not always prevent attacks. Anæmia and syphilitic infection of the central nervous system are two common associates of this form of syncope, and both may require energetic treatment.

(d) **Gowers' syndrome.** Often confusedly designated a "heart attack," this condition has for its leading symptom a sense of impending death associated with profound weakness and exhaustion, faintness, slight breathlessness, and tingling of the extremities. It lasts from 10 to 30 minutes. The patient, though distressed, does not lose consciousness and does not look seriously ill. The attack is probably a transient disorder of the autonomic nervous system and is commonly produced by prolonged mental strain or anxiety.

In treatment the first step is reassurance. Rest in bed is essential, usually for some days after the acute phase has subsided. Sedatives such as bromides 0.65 G. (gr. 10) three times a day or phenobarbitone 32 mg. (gr. $\frac{1}{2}$) twice or three times a day should be given. Particular emphasis deserves to be placed on the provision of adequate sleep. A rapidly acting barbiturate such as Cyclobarbitone B.P.C. 0.2 G. (gr. 3) (Phanodorm) followed by bromide 1.3 G. (gr. 20) is a useful and effective combination.

(e) **Stokes-Adams seizures** are syncopal attacks associated with a defect, transient or permanent, in the conducting mechanism of the heart beat. The milder attacks, in which the pause between successive ventricular contractions does not exceed six or eight seconds, cause no more than a momentary sense of insecurity and giddiness. A longer pause is followed by sudden loss of consciousness, and in the worst attacks, respiratory arrest and convulsions occur.

Recovery from the attack coincides with the resumption of the ventricular action. As a rule this occurs before any particular treatment can be given. In the most severe attacks, injections and inhalations are valueless, as the circulation is in abeyance. In desperate cases 0.2 ml. of Injection of Adrenaline B.P. may be given into the heart itself (*see pages 20 and 448*).

Ventricular action is restored immediately as shown by the flush of blood through the skin of the face. This is an emergency measure not without danger and is only justifiable for urgent situations such as a *severe* Stokes-Adams seizure, and the cardiac arrest of anæsthesia, electrocution, anaphylactic shock, drowning, and asphyxia neonatorum.

Rarely Stokes-Adams seizures recur at short intervals, the patient regaining consciousness only to relapse into coma every few minutes. In such circumstances, Injection of Adrenaline B.P. 0.5 ml. subcutaneously is a valuable remedy. The beneficial effect becomes apparent in 10 or 15 minutes, by which time the seizures may stop. If they persist the dose should be repeated.

Attacks occurring sporadically at longer intervals may be warded off by ephedrine 32 to 65 mg. (gr. $\frac{1}{2}$ to 1) by mouth every four to eight hours. It increases the excitability of the ventricular muscle. The dose should be no more than that necessary to increase the ventricular rate under resting conditions by three or four beats per minute. When ephedrine fails, a trial may be made with one of the slow acting adrenaline compounds (*page* 652) once or twice daily. Alternatively isoprenaline (Neo-epinine) 10 to 15 mg. sublingually every three or four hours may be helpful during the day and a single injection of slow acting adrenaline (*page* 652) at night. Atropine 1.3 to 2 mg. (gr. $\frac{1}{50}$ to $\frac{1}{33}$) subcutaneously is a remedy commonly employed. It is of little or no value.

Rarely recurrent Stokes-Adams seizures result from the transient interruption of co-ordinated ventricular contraction by short phases of ventricular fibrillation. This can only be recognised with certainty by the use of the electrocardiograph. It should be suspected when multiple seizures fail to respond to ephedrine or adrenaline. Theoretically when ventricular fibrillation is responsible for the Stokes-Adams attack it is justifiable to prescribe quinidine sulphate at four-hourly intervals but in my experience the seizures have been longer and more frequent when this drug was employed. Isoprenaline (Neo-epinine) sublingually and atropine subcutaneously are worth a trial.

• COLLAPSE AND SHOCK

There is no fundamental difference between "medical" collapse and "surgical" shock. The underlying mechanism is a decrease in the amount of blood in *active* circulation as a

result of disorganisation of the peripheral circulation. The ashen-grey colour, restlessness, exhaustion, cold damp skin, almost imperceptible pulse, shallow respirations and impaired mental acuity, make a characteristic picture often dramatic in its rapid development. As a medical emergency collapse is associated chiefly with acute infections such as lobar pneumonia (page 142) ; with profuse hæmorrhage from a bleeding peptic ulcer (page 85) ; or perhaps most commonly a myocardial infarct. The essential features common to all varieties are a reduction in the volume of circulating blood, a fall in blood pressure, impaired oxygenation of the tissues and a progressive bodily enfeeblement.

Treatment.—The classical remedies for shock are reassurance, posture, warmth, and morphine.

(a) REASSURANCE.—It is the duty of the physician to do what he can to counter the sense of apprehension and the fear of impending disaster—commonly associated with even minor grades of shock. Reassurance and encouragement mean much to shocked patients.

(b) POSTURE.—While the patient's condition is being assessed and preparations made for the major methods of treatment, attention should be devoted to nursing care. Posture is important. Unless otherwise contra-indicated, the patient should be encouraged to lie flat in bed, pillows and supports being withdrawn from the head. The foot of the bed should be raised on blocks, one to two feet high, to facilitate the return of blood to the heart and brain.

(c) WARMTH.—Since cold increases shock a generous supply of hot bottles, or the application of a "shock cage" in which heat is maintained by electric bulbs, is of value. The hands and feet must be kept warm. Similarly the provision of hot drinks is valuable in correcting dehydration and giving warmth. Hot soups, hot physiological saline with glucose, strong tea or coffee may all be administered with benefit.

(d) MORPHINE.—Pain is a potent factor in perpetuating those reflexes which disorganise the peripheral circulation and promote collapse. Morphine is well tolerated in the early stages and may be given in full doses hypodermically or intravenously. After some hours, when the disorder of the peripheral circulation is more advanced, the degree of shock more profound, and the patient

more enfeebled, smaller doses of morphine 8 to 11 mg. (gr. $\frac{1}{8}$ to $\frac{1}{6}$) are justifiable if only to reduce restlessness and give peace of mind. The detrimental effect of pain is to be dreaded more than the peripheral reaction to this drug, and there can be few instances in which morphine is not indicated for a shocked patient.

Other methods of treatment are valuable in special cases.—

(e) TRANSFUSION.—War experience has amply confirmed the view that the most effective therapeutic agent in counteracting the essential deficiency in shock or collapse is the restoration of an effective blood volume. When shock results from massive hæmorrhage, transfusion of whole blood is of prime importance. If suitable typed blood is not immediately available, various substitutes may be used, but whole blood is preferable. It may be given from a suitable container into a vein at a rate of 100 to 200 ml. per hour (*see page 48*) for 12 to 24 hours or even longer, depending on the patient's progress and general reaction. In assessing the situation, blood pressure readings are of the greatest importance. A fall in systolic pressure below 100 mm. is a danger signal and usually indicates the necessity for continued transfusion.

In the absence of matched blood, or when replacement by blood itself is not essential, the restoration of a satisfactory circulatory volume can be brought about by physiological saline, 5 per cent. dextrose in physiological saline, or reconstituted mixed plasma. These agents lack the oxygen-carrying capacity of blood, and while saline and dextrose are helpful in an emergency, they do not provide the colloid osmotic pressure of plasma which maintains fluid within the circulation. Dextran—a glucose polymer with a molecular weight conforming to that of albumin—is not pyrogenic, antigenic or toxic and is said to produce a prompt and lasting increase in blood pressure. It is given slowly by intravenous drip as a 6 per cent. solution in physiological saline.

(f) OXYGEN. (*see page 569*)—Of the various remedies used in the treatment of circulatory collapse, oxygen is secondary in importance only to morphine. Anoxæmia has a detrimental effect on both the central and vegetative nervous systems and thus aggravates all the features of shock. It is of particular value in those forms of anoxæmia in which oedema of the lung alveoli

interferes with the absorption of oxygen. Pulmonary and myocardial infarction are the two conditions in which oxygen is of particular value whether cyanosis be present or not. .

- (g) ANALEPTICS.—Synthetic compounds such as nikethamide or leptazol (*page 608*) are useful and effective remedies for cardio-respiratory failure in doses of 1 ml. or more, intramuscularly at one or two-hourly intervals. Much larger doses (10 ml. in 250 ml. of physiological saline by intravenous drip) may be used in circulatory collapse when the critical situation justifies desperate remedies. It is doubtful if either of these drugs is active by mouth. They may be used alternately with strychnine 2 mg. (gr. $\frac{1}{32}$) by two-hourly intramuscular injections over the acute phase of respiratory failure in pneumonia, septic peritonitis or acute delirium tremens. They are also useful for the mild degree of collapse inseparable from full and repeated morphine administration.

(h) EXTRACT OF SUPRA-RENAL CORTEX.—While the ideal drug for the treatment of acute peripheral circulatory failure has not yet been found, Injection of Supra-renal Cortex B.P.C., by correcting the abnormal capillary and cellular permeability, is believed to control the volume of circulating fluid. It has been reported useful in the prevention of experimental shock and its great value in the shock of acute adrenal cortical insufficiency is the main justification for its employment in other forms. It may be added to the transfusion fluid in the proportion of 2 ml. to each litre.

Remedies of doubtful value.—Digitalis is contra-indicated in acute peripheral circulatory failure since it accentuates shock by lowering venous pressure and so further diminishes cardiac output. Similarly there is little or no justification for the use of adrenaline or pituitary extracts (vasopressin).

PALPITATION

This may result from extrasystoles or from paroxysmal tachycardia.

Extrasystoles in nervous subjects may cause alarm and fatigue and give rise to an urgent call. Less sensitive subjects are often unaware of the irregularity. Extrasystoles are often easily

recognised as they only interrupt a rhythm which is fundamentally normal and are abolished by exercise. Symptoms can be alleviated by bromide or phenobarbitone. If the irregularity persists and disturbs sleep, quinidine should be tried, giving 0.1, to 0.3 G. in a capsule, at 4 p.m. and 9 p.m. Otherwise, the drug should be taken an hour or two before the disturbance usually occurs.

PAROXYSMAL TACHYCARDIA

This implies abnormally rapid rhythm of abrupt onset and usually self-limited in duration. As a rule the patient is immediately aware of the disturbance, the vigour of the heart's action contrasting sharply with the feebleness of the pulse. The abnormal mechanism is largely outside nervous control and hence the rate shows little or no variation with respiration, change of posture, or muscular activity. It keeps remarkably constant from minute to minute, hour to hour, and indeed from day to day. This is in sharp contrast to simple tachycardia in which the rate is susceptible to such simple activities as sitting up in bed or taking a few deep breaths.

From the point of view of treatment, it is important to determine the nature of a paroxysm of tachycardia. The chief features of the three important varieties are shown in the table (Fig. 18).

Although there are exceptions to each point stressed in the table, yet systematic consideration of these items usually makes the diagnosis clear. The important points may be emphasised as follows. Rates of 180 or more are unlikely to result from tachycardias other than those of the paroxysmal variety. In elderly patients the ventricular type is probable if coronary disease is also present. Auricular paroxysmal tachycardia is a more innocent but recurrent condition affecting younger people. Carotid sinus pressure is particularly helpful in the recognition of flutter since it causes a temporary disturbance of the rate, the ventricles slowing and becoming irregular for a few seconds before the fast regular rhythm is resumed. Neither paroxysmal auricular tachycardia nor ventricular tachycardia react in this way. Paroxysmal auricular fibrillation seldom causes confusion, the totally irregular action of the ventricles being so characteristic. The diagnosis can often be made with certainty, therefore, on

	<i>Auricular tachycardia.</i>		<i>Auricular flutter.</i>	<i>Ventricular tachycardia.</i>	<i>Remarks.</i>
Age at onset.	Usually before age 40.	Usually after age 40.	Generally after age 40.	Generally after age 50.	•
Ventricular rate	Usually above 160.	Usually below 160.	Usually below 160.	Usually above 180.	Higher rates are much in favour of auricular and ventricular tachycardia and against flutter.
Ventricular rhythm.	Regular.	Regular but occasional transient interruption for a cycle or two sometimes occurs.	Regular but occasional transient interruption for a cycle or two sometimes occurs.	Very slight arrhythmia can sometimes be detected on auscultation.	On account of a pulse deficit, rate and rhythm should be estimated by auscultation.
Duration of the attack.	1-2 hours or less. 2-3 days at the most.	A day or two as a rule.	A day or two as a rule.	Several days or even a week or two.	
Previous attacks.	Very frequent.	Common.	Common.	Exceptional.	
Carotid sinus stimulation.	May abruptly abolish the attack or have no effect.	A temporary irregular ventricular rhythm induced. Tachycardia returns immediately after transient slowing.	A temporary irregular ventricular rhythm induced. Tachycardia returns immediately after transient slowing.	No influence on rate or rhythm.	Right or left carotid sinus stimulation may be employed.
Underlying state of heart muscle.	Commonly healthy.	Usually damaged but relatively slightly.	Usually damaged but relatively slightly.	Gross damage, severe in degree, generally present.	
Associated conditions.	Mitral stenosis is fairly frequent.	Coronary sclerosis common.	Coronary sclerosis common.	1. Recent myocardial infarction. 2. Over digitalisation in congestive heart failure.	Ventricular tachycardia exceptional in otherwise healthy heart, but does occasionally occur.
Effect of digitalis.	May abolish abruptly.	Causes ventricular irregularity and slowing. Induces auricular fibrillation.	Causes ventricular irregularity and slowing. Induces auricular fibrillation.	Aggravates, and is contra-indicated.	

clinical grounds, but the help of the electrocardiograph should not be omitted.

Paroxysmal auricular tachycardia is characterised by the abrupt onset of a rapid regular heart rate of 160 to 220 beats per minute. The patient complains of a fluttering feeling in the chest and grows exhausted, with an urgent desire to lie down. In the absence of organic heart disease symptoms may be no more than faintness or weakness, as attacks are usually short-lived. In older people, and when complicating heart disease, paroxysmal tachycardia can cause considerable distress, overburden the heart, and induce congestive heart failure.

Treatment.

(i) **REFLEX VAGAL STIMULATION.**—Patients often discover for themselves some simple manœuvre on which they rely to cut short the attack. Holding a long deep breath, powerful expiration against a closed glottis, and firm pressure on the abdomen are such methods. They are more likely to be successful if used early in the attack.

(ii) **CAROTID SINUS PRESSURE.**—This is more effective when the patient is sitting than when lying down. Face the patient and place the fingers of the left hand behind the neck, and the thumb just below the angle of the jaw on the right side. Press the carotid artery firmly backwards against the vertebrae with the thumb. It may be necessary to obliterate the carotid lumen for 10 to 20 seconds. If pressure on the right side is ineffective after two or three trials at intervals of a few minutes, the same technique may be employed on the left side, and a short application may even be made on both sides simultaneously. Pressure on an eyeball seldom succeeds but may be tried if the patient will tolerate it.

(iii) **SEDATIVES.**—Attacks usually cease spontaneously. If they do not, or if the above measures fail, a dose of chloral hydrate 2 G. (gr. 30) will often prove helpful—the patient waking after a refreshing sleep to find that normal rhythm has returned.

(iv) **REFLEX EMETICS.**—Fairly intense vagal stimulation by means of reflex emetics is sometimes effective in terminating a paroxysm. This method is employed in patients with organic heart disease where delay might be harmful, or in others when the paroxysm has lasted many hours and the patient shows signs of exhaustion.

Tincture of ipecacuanha is given, a teaspoonful at a time, every five minutes until vomiting occurs. Up to six doses may be needed.

•(v) PARASYMPATHETIC STIMULANTS.—Similar, but much more powerful, vagal stimulation can be induced by the acetyl-choline group of drugs. The more stable compounds suitable for subcutaneous use are Methacholine chloride B.P. (Mecholyl; acetyl- β -methylcholine) and Carbachol B.P. (Moryl, formerly called Dóryl). For younger people the dose of Mecholyl is 15 to 30 mg., and for patients over fifty, 30 to 50 mg. subcutaneously (not intravenously). If Mecholyl is ineffective, carotid sinus pressure should be re-tried since it is more likely to be successful after Mecholyl. Side effects such as flushing, sweating, an abrupt bowel action, vomiting, exhaustion, and collapse commonly follow from parasympathetic stimulation. These drugs should therefore be used with caution. The patient should be recumbent, and it is wise to have a bed pan at hand and a syringe already charged with 1 to 2 mg. (gr. $\frac{1}{64}$ to $\frac{1}{32}$) atropine sulphate *at the bedside* before a parasympathetic stimulant is used. Atropine counteracts the vagal effects and should be administered intravenously on the first appearance of any disagreeable symptom. Further absorption of the choline compound can be retarded by the light application of a tourniquet to the limb, and by the application of ice at the site of the injection.

(vi) DIGOXIN.—In my experience, digoxin, with certain provisos, is a safer and more reliable remedy than the choline compounds. It should not be given in a full dose to any patient who has taken digitalis or a related substance within the preceding 10 days. Further, it is desirable to be certain of the nature of the paroxysm before digoxin is used. This necessitates electrocardiography. If it is known that the paroxysm is *not* of the ventricular variety, digoxin can be employed with justification. Given intravenously in a dose of 1.0 to 1.5 mg. suitably diluted in 10 or 20 ml. of physiological saline, digoxin by its vagal stimulant properties is capable of arresting auricular tachycardia often within 10 or 20 minutes.

(vii) PROCAINE AMIDE (Pronestyl hydrochloride. Squibb).—If the arrhythmia persists despite these measures 250 to 500 mg. of

procaine amide in 50 to 100 ml. of sterile water or saline may be given slowly into a vein.

(viii) **QUINIDINE**.—If procaine amide fails and the patient's condition is deteriorating, quinidine may be tried. The oral route is preferable, using quinidine sulphate in capsules or cachets in doses of 0.2 to 0.4 G. at four-hourly intervals. Alternatively, quinidine lactate, 0.3 G. in 30 ml. saline, may be *slowly* injected intravenously, preferably under electrocardiographic control.

Paroxysmal ventricular tachycardia is usually associated with organic heart disease. It may be an alarming and serious complication of acute myocardial infarction, or may arise as a manifestation of digitalis intoxication in elderly patients.

Treatment.—The first step is to give a sedative—bromide, barbiturate, or morphine, depending on the urgency and duration of the symptoms. Ipecacuanha, Mecholyl, and digoxin, and mechanical methods of vagal stimulation are valueless and contra-indicated. Until recently quinidine has been the drug of choice but procaine amide (Pronestyl hydrochloride, Squibb) is prompt and comparatively safe. A dose of 0.5 to 1.0 G. (*i.e.*, 5 to 10 ml. of a solution containing 100 mg. per ml.) can be given intramuscularly and repeated at two to three-hourly intervals or half this amount may be given by slow intravenous drip.

If it is decided to use quinidine a suitable scheme is to give 0.2 G. quinidine sulphate by mouth at four-hourly intervals with the omission of the 2 a.m. dose so as not to disturb sleep. The dose can be continued safely for 36 to 48 hours if the attack persists. Larger doses can be used, preferably under electrocardiographic control. Usually the ventricular rate slows progressively under the influence of quinidine until it is around 90 to 110, when the abnormal rhythm ceases abruptly.

When conditions are more urgent, the patient's distress increasing, and portal and peripheral congestion complicating the situation, it may be considered expedient to use quinidine intravenously, preferably by continuous intravenous drip. Too rapid injection may cause serious circulatory collapse. By vigorous shaking, 3 to 4 grammes of quinidine sulphate can be dissolved in 500 ml. of a 5 per cent. dextrose solution. After filtering and warming, the sterile solution is run into a vein at a rate of 1 to 2 ml. per minute until normal rhythm returns, or until cinchonism shown by headache, roaring in the ears, and some degree of

deafness is induced. In my experience, this is a reasonably safe and effective method of administering quinidine sulphate intravenously. It seldom fails to restore normal rhythm. Quinidine lactate is said to be less liable to cause toxic effects. A dose of 0.65 G. (gr. 10) is diluted in 50 to 150 ml. of 5 per cent. dextrose and given intravenously at 2 ml. per minute.

Paroxysmal auricular flutter resembles paroxysmal auricular tachycardia, and may be distinguished by the temporary slowing and irregular action induced by carotid sinus stimulation. It is probably the rarest of the acute paroxysmal disorders and gives rise to less trouble in that the heart rate is seldom over 160 beats a minute, and individual attacks are of short duration.

Treatment.—Digitalis is indicated. It is seldom necessary³ to use the intravenous route, but if the rhythm causes distress and attacks occur with sufficient frequency, it is wise to allow the patient to have a supply of a reliable digitalis preparation such as digoxin, which can be taken in a full dose by mouth at the onset of the attack. Providing that none has been taken during the preceding week, 1.0 to 1.5 mg. (i.e., up to six 0.25 mg. tablets) can be taken at once. Dried digitalis leaf is equally, if not more, efficient but, as it is excreted more slowly than digoxin, full dosage must not be given until two weeks have elapsed since the last dose of digitalis, so as to avoid intoxication. Rapid digitalis effects may be achieved by a full dose of 1 to 1.3 G. (gr. 15 to 20) of the powdered leaf in divided amounts over a 24-hour period. Digitalis breaks up the regular ventricular rhythm of auricular flutter, converts flutter to fibrillation, and may bring about a reversion to normal rhythm. On account of the danger of doubling the ventricular rate by the abolition of block, quinidine is better not used in the treatment of paroxysmal flutter.

Paroxysmal auricular fibrillation.—This occurs more commonly than auricular flutter. After operation in elderly people, in the course of acute infections such as pneumonia, and particularly in thyrotoxicosis, bouts of auricular fibrillation are common. Distress results from the excessive rate, the thumping irregular beats, and occasionally from a choking sensation in the throat.

Treatment.—When subjective distress is obvious, or when signs of heart failure appear imminent, treatment with digitalis is desirable. The object is to bring the grossly excessive rate

under control. This can be done either by using digoxin or digitalis leaf as described for auricular flutter. The heart rhythm usually reverts to normal within a few days of digitalisation. Quinidine is reserved for those patients in whom digitalis fails. When paroxysmal fibrillation occurs after valvotomy it is an indication for anticoagulant therapy with the object of preventing the thrombo-embolism otherwise prone to occur.

DYSPNŒA

(a) **Acute left ventricular failure.**—Extreme dyspnœa occurring at rest, often during the night, perhaps after a strenuous day, is a symptom of considerable consequence (*see also page 118*). The early stages of the attack are sometimes spoken of as “cardiac asthma” and the late stages which treatment is designed to avoid as “acute pulmonary œdema.” Occurring in a paroxysmal form, and preceded by a persistent tickling cough, with increasing restlessness, it is a sign of a failing left ventricle, commonly the result of long continued hypertension, aortic stenosis or insufficiency, a myocardial infarct, or less commonly a tight mitral stenosis. Unless relieved, the urgent dyspnœa advances to acute pulmonary œdema with the production of white, and later pink, frothy sputum. Such a condition arises primarily as a result of a disproportion in the ventricular outputs, the left being unable to deal with all the blood fed into the pulmonary circuit by the right side of the heart. In these circumstances it has been said that the patient “tends to drown in his own sputum.”

Treatment.—The objects of treatment are to allay restlessness, increase the efficiency of the left ventricle, and to reduce temporarily the output of the right ventricle.

The patient prefers to sit bolt-upright, supported by pillows, with the feet dependent. He must be kept warm, reassured, and given morphine 16 to 32 mg. (gr. $\frac{1}{4}$ to $\frac{1}{2}$) intramuscularly at once. By this means his strength is conserved, the exhausting cough relieved, the venous return reduced, and dyspnœa eased.

When attacks are severe and the response to the above measures unsatisfactory, a full dose of digitalis should be administered, provided that no allied preparation has been taken in therapeutic amounts in the preceding two weeks. For a prompt effect there is no better preparation than digoxin 1.0 to 1.5 mg.

diluted in 10 to 20 ml. of physiological saline by the intravenous route.

Venesection is the next step (*page 580*) and, in the presence of white or pink frothy sputum there should be no hesitation in performing it. A "bloodless phlebotomy" can be performed by the application of the sphygmomanometer cuffs, bandages, tourniquets, or towels to the proximal portion of the thighs and arms at a pressure only a little in excess of the diastolic blood pressure. In this way blood may be temporarily shunted from the right side of the heart. Compression of the limbs is continued from 10 to 20 minutes and then gradually released from one extremity after another at intervals of a few minutes. The whole procedure can be repeated if necessary. With similar intent, the administration of nitroglycerine has been recommended. A tablet of glyceryl trinitrate 0.65 mg. (gr. $\frac{1}{100}$) allowed to dissolve under the tongue causes dilatation of the splanchnic vessels and temporarily reduces the venous return to the heart, and theoretically should lessen the right ventricular output. As soon as practicable, oxygen should be administered.

Aminophylline (*see page 598*) 0.25 to 0.5 G. (= 10 to 20 ml.) by slow intravenous injection may be used with advantage if Cheyne-Stokes respiration complicates the acute phase of the illness. Diuretics are helpful in the prevention and treatment of acute left ventricular failure but their effect is delayed for some hours. Mersalyl B.P. 1 to 2 ml., or Neptal 1 to 2 ml. may be given intramuscularly (*page 42*). A specially prepared solution of Neptal may be used intravenously in doses of 5 ml. These are potent agents in the treatment of left ventricular failure but some hours elapse before real benefit is obtained from their use.

(b) Acute right ventricular failure results from acute dilatation of the pulmonary artery and right ventricle caused most commonly by the impaction there of a massive embolus' (*see also page 133*). When this is very large and obstructs one or other pulmonary artery, death results almost instantaneously. In less severe instances there is intense dyspnoea, thoracic oppression, substernal pain and the rapid development of circulatory collapse, the whole picture bearing a striking resemblance to coronary thrombosis from which diagnosis may be difficult (*see page 133*).

Morphine should be used liberally, 11 to 16 mg. (gr. $\frac{1}{8}$ to $\frac{1}{4}$) intramuscularly, and oxygen administration started at once. By

this means dyspnoea, cyanosis, pain and anxiety can be eased appreciably and shock lessened. Venesection is to be condemned as it depletes further a circulation already hampered by the disorganisation of peripheral circulatory failure. Indeed, a falling blood pressure might be taken as an indication for a dextrose saline transfusion. Nikethamide or leptazol by subcutaneous injection at two-hourly intervals in a dose of 1.0 to 1.5 ml. is often helpful in warding off the harmful effects of a profound degree of collapse which can develop rapidly. Atropine 0.65 mg. (gr. $\frac{1}{100}$) combined with papaverine hydrochloride, 32 to 50 mg. (gr. $\frac{1}{2}$ to $\frac{3}{4}$) by the intravenous route, is said to ease the situation by the relaxation of spasm in the pulmonary and coronary vessels. Finally, the heroic procedure of embolectomy has been attempted with success in rare instances.

(c) Cardiac tamponade.—This term is applied to the picture which results from an increase in intra-pericardial pressure sufficient to interfere with the venous return to the heart. The combination of pulsus paradoxus (a pulse which diminishes in force or even vanishes during inspiration) with pulsating neck veins is an important diagnostic clue. Excessive amounts of fluid within the pericardium may accumulate rapidly, and, unless the pericardial sac stretches sufficiently, the great veins are compressed, the venous inflow retarded, and the ventricular outputs reduced. A mechanical reduction of the intra-pericardial pressure may be considered desirable during the course of rheumatic pericarditis if it be judged that the effusion is accumulating with such rapidity as to lead to venous engorgement in the neck, and a progressive fall in blood pressure. It is said that the best indication for withdrawal of pericardial fluid is a pulse pressure below 20 mm. Day to day blood pressure records in pericarditis with effusion are of considerable value. Tapping the pericardium is a procedure only to be undertaken as a last resort when orthopnoea is severe, thoracic and epigastric oppression a real embarrassment, and when the pulse pressure is falling steadily. Most cases of pericardial effusion of rheumatic origin make a good recovery without tapping. It is more often necessary in tuberculous or malignant pericarditis, septic infection of the sac, or on account of bleeding from wounds of the heart.

In rheumatic pericarditis with a large effusion, a safe measure is to puncture the pericardium from behind (*page 530*).

PRÆCORDIAL PAIN

Effort Angina and Myocardial Infarction.—The clinical picture of acute myocardial infarction is well known—prolonged pain, some degree of shock, followed by a minor fever, a leucocytosis and a disturbed sedimentation rate being the outstanding features. There are all grades of severity so that on occasion it may be difficult to distinguish the severer attack of angina pectoris from a minor myocardial infarct. If the first attack of angina of effort is sufficiently severe to warrant an emergency call, it is justifiable to assume that an acute myocardial infarct has been sustained. Similarly, should anginal attacks in a known subject of coronary disease suddenly increase in frequency or severity and particularly if pain of a genuine anginal character is experienced at rest—"angina decubitus"—the doctor should suspect that a local thrombosis is imminent, if indeed it has not already occurred. In these circumstances treatment should be as for acute myocardial infarction.

Treatment.—The immediate objectives are to relieve pain, to counteract shock, to limit the size of the resulting infarct, to reduce the risks of complications and to promote a firm healing of the infarcted area.

MORPHINE.—Complete bed rest reduces myocardial activity to a minimum, tends to limit the area infarcted, conserves energy and favours healing. It is therefore a first essential. Morphine 16 to 32 mg. (gr. $\frac{1}{4}$ to $\frac{1}{2}$), repeated in sufficient quantity to abolish pain, thereby lessens shock and allays anxiety. Unless dyspnoea is troublesome at the onset, when the shoulders may have to be supported, the patient should be put to bed at once and the head raised on one pillow. Warmth should be maintained by hot bottles and hot drinks. Over-sedation is to be avoided. Morphine in full doses may accentuate shock by causing excessive sweating and persistent vomiting.

HOME OR HOSPITAL?—In the knowledge that the efficient use of anti-coagulants has halved the death rate for the first six weeks after an acute myocardial infarct, the practitioner will have to decide on the advisability of moving the patient to hospital. This depends on a number of factors. If a hospital equipped for the laboratory control of clotting times is within easy reach, then there is much to be said for moving the patient there at the outset

rather than some days later when his condition may be more precarious. If the doctor knows that the laboratory facilities in the neighbourhood are such that he himself can accept the responsibility of using anticoagulant therapy and all that it entails, then there is less justification for advising the hospital treatment. With few exceptions, the doctor is wise to assume at the outset of the illness that energetic measures for the prevention of those recurrences and complications responsible for the high mortality rate will be necessary. The hospital's resources are directed to the correction of shock, the prevention of the thrombo-embolic complications and the avoidance of congestive heart failure, which are the main causes of death within the first six weeks.

SHOCK.—The severer grades of cardiogenic shock are associated with an 80 per cent. mortality and active measures for their prevention and treatment are highly desirable. The ideal drug with which to counter the ill-effects of acute hypotension is not yet known, but leptazol 1·0 ml. intramuscularly, ephedrine 25 to 30 mg. intramuscularly at intervals of two to three hours, and Methylamphetamine hydrochloride B.P.C. (Methedrine) 5 mg. intravenously, with a sustaining intramuscular injection of 10 mg. repeated if necessary at three hourly intervals are all of value.

OXYGEN.—Oxygen, at 6 to 7 litres per minute (*page* 569), is helpful over the first 24 or 48 hours. It is often advisable to arrange for its use in the ambulance, if there is a trained nurse or a first-aid attendant familiar with its administration.

INTRAVENOUS AND INTRA-ARTERIAL TRANSFUSIONS.—These have not so far yielded encouraging results in the treatment of cardiogenic shock.

DIGITALIS.—When the severer grades are accompanied by orthopnoea and pulmonary oedema, even of milder degree, the use of a digitalis preparation can be readily justified. For this purpose digoxin 1·0 mg., diluted with 10 ml. of saline, intravenously, may help to sustain the uninfarcted muscle, improve coronary flow and reduce the added embarrassment of left ventricular failure superimposed on the shock syndrome. Digoxin is not a drug for routine use in acute myocardial infarction but it is wise to use it on the first indication of left ventricular failure.

ANTI-COAGULANTS.—In most cases, as soon as the diagnosis is established, the doctor should administer heparin, 10 to 15 thousand units (*page* 595) intravenously at once, even before

sending the patient to hospital. There is good evidence to suggest that the sooner anti-coagulants are used, the better is the outlook. By their immediate use the formation of intra-ventricular and other clots, with the subsequent dangers of their detachment, can be lessened. Moreover, it appears that the prompt use of anti-coagulants has a favourable influence on the outcome of even the severest grades of cardiogenic shock. Heparin is a comparatively safe anti-coagulant and its use in the early stages of the illness is to be encouraged.

In the hospital a convenient routine is to administer heparin intramuscularly in doses of 20,000 units at six to eight hour intervals. It is usually only necessary to continue heparin for 24 to 48 hours, by which time the oral anti-coagulant, phenindione (Dindevan) or nicoumalone (Sinthrome) (*page 596*) started simultaneously with heparin, has generally induced a sufficient prolongation of the prothrombin clotting time.

The haphazard use of anti-coagulants without laboratory control cannot be too strongly condemned. Although the practitioner can begin their use in the patient's own home, these drugs should not be continued beyond the first 48 hours unless reliable laboratory facilities are available. Hæmorrhage is the main danger in their use and this can only be avoided by regulating the dose according to the prothrombin time (*page 596*).

PERIPHERAL EMBOLISM

Most arterial emboli arise from the heart, thrombi becoming detached from the valves, as in bacterial endocarditis, or from the endocardium as in mitral stenosis or myocardial infarction. The lodgment of a clot in a peripheral artery obstructs the blood supply, often produces an intense vascular spasm throughout the limb distal to the clot, and gives rise to local anoxæmia manifesting itself by numbness and coldness of the extremity, a more or less cadaveric appearance of the limb, and often by considerable pain throughout the affected area. The oft-quoted alliterative description of symptoms—"pain, pallor and paralysis" is somewhat misleading since acute pain occurs at the onset in perhaps only 50 per cent. of cases. If the local circulatory deficiency be not made good by adequate collateral channels, or by the timely removal of the clot from the major vessel, the tissues perish and at a level varying with the site

of the arterial block, a line of demarcation forms, distal to which, the limb becomes gangrenous.

In the arm, on account of the richer blood supply, embolectomy is seldom required, but when a leg is affected it is wise to assume at the outset that surgical removal of the clot will be necessary within an hour or two. For operative success, the general condition of the patient must warrant interference and the procedure must be undertaken within 10 hours of impaction of the clot. Experience has shown that the longer the embolus remains impacted, the greater the local injury to the arterial wall, and that even after its removal, thrombosis on the damaged intima is prone to occur and may spread peripherally. Prompt measures can often save the limb. Allen, from a large experience, emphasises three important "don'ts." Don't delay, don't elevate the limb, and don't overheat it.

DON'T DELAY.—Conservative measures are important, not only when an operation is contra-indicated, but also when surgery cannot be immediately undertaken. Indeed, the medical measures now advocated may prove so satisfactory that surgical help may not be necessary, but a decision must not be postponed too long. If appropriate medical measures have not produced a *striking* improvement in the state of the limb *within three or four hours of the lodgment of the clot*, then embolectomy should be seriously considered.

DON'T ELEVATE THE LIMB.—The object in treatment is to increase the blood supply. This can be done mechanically by elevating the head of the bed on blocks when the leg is involved, or, in the case of the arm, by keeping it dependent.

DON'T OVERHEAT THE LIMB.—Heat is essential but should not be applied directly to the limb. It is a common mistake to surround it with hot water bottles. By increasing the local metabolism, excessive heat accentuates the nutritional deficiencies, aggravates the tendency to gangrene, and commonly provokes extensive burns of the devitalised skin. The entire limb should be wrapped in warm cotton wool held lightly in place by a bandage, thus preserving its natural temperature and protecting it from the ill effects of excessive heat. A cradle, preferably left open at one end, and containing one, or not more than two, light bulbs, may be placed over the limb, but the temperature of the air within the cage should not exceed 105°F. (40.5°C.).

Learmonth has shown how effective reflex vaso-dilatation may be in increasing the blood supply. By heating the hands, blood-flow in the foot and leg may be enhanced. A warm bed jacket should be worn to protect the arms from chilling, and with one hand in a box, or under a cradle, heated by a light bulb, the temperature may be raised sufficiently high to induce local sweating. A glove should be worn on the opposite hand. By this means vaso-constriction in the affected foot and leg may be released reflexly.

If pain is severe, morphine 11 to 16 mg. (gr. $\frac{1}{6}$ to $\frac{1}{4}$) should be given hypodermically. Less intense pain can be controlled by a compound tablet of aspirin, phenacetin and codeine taken by mouth as necessary. Immediately the diagnosis of embolism is made 10,000 units of heparin (*see page 595*) should be given intravenously provided anti-coagulants are not already in use. The object is to prevent extension of the thrombus. Heparin should be repeated four to six-hourly until embolectomy is performed or the fate of the limb decided. Papaverine hydrochloride has valuable anti-spasmodic properties and is of great help when given intravenously in a dose of 32 to 50 mg. (gr. $\frac{1}{2}$ to $\frac{3}{4}$). Alcohol, in the form of whisky or brandy, is a useful vasodilator when taken by mouth, and may therefore be prescribed generously. Tolazoline (Priscol, Ciba) 25 to 50 mg. may be given intravenously to reduce peripheral vaso-constriction. If benefit is obtained a similar quantity can be taken by mouth at 4 to 6 hour intervals. The side effects are chiefly nausea and abdominal pain but sensations of coldness and palpitation also occur. Spinal anæsthesia is a further procedure which can be used to promote maximal but temporary vaso-dilatation when the leg is involved.

If it be judged that the patient is unfit for embolectomy, or if more than 10 or 12 hours have elapsed since the embolism, the area of the limb liable to the development of gangrene must be kept clean and dry by the application of surgical spirit and sterile dusting powder once or twice daily. Oral anticoagulants should be given for two or three weeks. Amputation can be undertaken at a later date, if the patient survives.

A. RAE GILCHRIST.

CHAPTER IX

Emergencies in Blood Diseases

WHEN it is suggested that a blood disease may be responsible for urgent symptoms, it is of the greatest importance that at least a provisional diagnosis should be made before commencing treatment, no matter how acutely ill the patient may be. Inappropriate treatment may waste time and, particularly in macrocytic anæmias, render the diagnosis more difficult. Although the history is suggestive in some cases, the symptoms are often too varied to be more than pointers to possible diagnoses.

A full "blood count" should therefore be made and should include the following procedures:—

- (a) hæmoglobin estimation,
- (b) erythrocyte count,
- (c) leucocyte count,
- (d) examination of a blood film and differential white cell count,
- (e) reticulocyte and platelet counts.

Bone marrow examination should be included if indicated.

In severe anæmia and in all cases where the condition has been caused by hæmorrhage, it is advisable to determine the patient's blood group as soon as possible and put in hand all necessary arrangements for carrying out transfusion at short notice.

ANÆMIAS

In **post-hæmorrhagic anæmias** the diagnosis is suggested by the history of hæmorrhage and the general symptoms of anæmia. The main hæmatological findings are a reduction in hæmoglobin and red cells. Hence after an acute blood loss there is at first a total reduction of all blood constituents with a normal hæmoglobin percentage, red cell count and colour index owing to loss of whole blood. Soon, however, with the subsequent hæmo-dilution by fluid withdrawn from the tissues, an anæmia develops with a low red cell count and much reduced hæmoglobin percentage. With chronic blood loss, red cell formation may outstrip the available hæmoglobin leading to a hypochromic microcytic anæmia, and the colour index may be very low (between 0.3 and 0.6). Sometimes there is also leucopenia. Bone

marrow examination may be very helpful in the diagnosis of anæmia secondary to malignant metastases, multiple myeloma and such conditions as Gaucher's disease.

• *Treatment.*—Bleeding should be stopped, if practicable, by appropriate measures including coagulant remedies (page 124) and adequate rest and warmth must be secured (see *epistaxis*, page 123; *hæmophilia*, page 180; *hæmatemesis*, page 85; *hæmoptysis*, page 126).

If the anæmia is severe (red cells less than 2,000,000 per cu.mm., hæmoglobin less than 50 per cent.) and in order to combat shock from severe blood loss, transfusions (at least two to three pints of whole blood) should be given as soon as possible to restore blood volume and raise the falling hæmoglobin concentration and red cell count. In all cases blood of the appropriate ABO and Rh. groups must be given after careful cross matching. If there is much reduction of blood volume with low blood pressure and dehydration, it may be necessary to give at once plasma or a plasma substitute or dextrose saline *after* blood has been taken for grouping purposes and while suitably cross-matched blood is being obtained. Care should be exercised with plasma substitutes which in some cases may be antigenic.

Aplastic anæmia.

In this condition the patient is often seen for the first time, acutely ill and with a fairly short history of increasing weakness, tiredness, dyspnœa, palpitation, disinclination to do anything, and marked waxy pallor of the skin. There has often been an exacerbation in the previous few days or weeks with bruising of the skin and bleeding from the gums, nose and mucous membranes and into the retinae and elsewhere.

The disease usually comes on very insidiously between the ages of 15 and 40 as a progressively severe normocytic normochromic anæmia without evidence of regeneration or abnormal cells in the peripheral blood, and a negligible reticulocyte count. Sternal biopsy shows severe hypoplasia or complete aplasia of the bone marrow. The spleen is rarely enlarged. The secondary types of aplastic anæmias come on at any age after X-ray therapy and exposure to toxic materials such as benzene, radio-active substances, gold, arsenic, sulphonamides, thiouracil, amidopyrine, chloramphenicol, etc., or as a sequel to severe overwhelming infections or sepsis.

Treatment.—The patient must be removed from any possible toxic influences. Repeated transfusions of fresh whole blood or preferably packed red cells of the correct ABO and Rh. groups will be required in large amounts. In a grave emergency in males the Rh. group may appear to be less important but it becomes of major significance in subsequent transfusions and so the blood must always be very carefully cross-matched. Much help can be given by the Regional Transfusion Officer.

Every endeavour should be made to raise the blood count to nearly normal as soon as possible since this gives longer periods of remission. Transfusion usually stops any bleeding from mucous surfaces. Many other methods of treatment have been tried, such as giving large doses of liver, iron, extracts of spleen, yellow and red bone marrow, folic acid, vitamin B₁₂, cortisone, corticotrophin, prednisone and prednisolone, but they are of no value.

Measures should be taken to minimise the special danger of infection to which these patients are liable by giving suitable antibiotics such as penicillin. Sulphonamides and chloramphenicol should be avoided.

Macrocytic anæmias are characterised by lower red cell counts relative to the hæmoglobin percentage, and a preponderance of large cells, with consequently a raised colour index. Sternal marrow biopsy discloses a typical megaloblastic hyperplasia. The clinical findings vary somewhat according to the place in the hæmopoietic mechanism at which the fault has occurred. **Pernicious anæmia** is the commonest type in temperate climates and quite often presents as an acute emergency judging from hospital experience.

Treatment of uncomplicated pernicious anæmia is relatively easy. A suitable preparation of cyanocobalamin (vitamin B₁₂) (Anacobin, British Drug Houses; Bitevan, Evans; Cytamen, Glaxo) should be given in doses of 100 to 300 microgrammes subcutaneously or intramuscularly three times in the first week and weekly thereafter. If the patient is oedematous the intravenous route is best. Oral preparations are not quick enough or certain enough for emergency treatment. Since a few cases may not respond very well to cyanocobalamin, some prefer to use potent liver extracts.

There is no place for blood transfusions in the treatment of patients suffering from pernicious anæmia, even in a grave emer-

gency for they often lead to fatal results. Repeated transfusions, however, are necessary in macrocytic anæmias of the achrestic type and in those associated with hepatic cirrhosis, since they are resistant to other treatment.

There are no indications for the use of folic acid in the treatment of pernicious anæmia. Only in non-Addisonian macrocytic anæmia such as the megaloblastic macrocytic anæmia of steatorrhœa or the macrocytic anæmia of pregnancy should folic acid be used (100 mg. parenterally). In these conditions it is more effective than vitamin B₁₂ or liver extracts.

Hypochromic microcytic anæmia and the Patterson-Kelly syndrome (dysphagia with achlorhydric anæmia) occur most commonly in women between 20 and 50. A similar anæmia, however, may be seen in either sex as a secondary manifestation of malignant disease, tuberculosis, nephritis, or after gastro-enterostomy and gastrectomy.

This type of anæmia usually develops insidiously and so the patient may not be seen until late in the course of the disease when some symptom such as excessive tiredness, exhaustion, pallor, dysphagia, loss of appetite and wasting makes her seek advice urgently. Sometimes anæmia with hæmoglobin values below 20 per cent. (2.96 G. per 100 ml.) appears suddenly.

Other symptoms of help in diagnosis are flatulent dyspepsia, intermittent diarrhœa, angular stomatitis, soreness of the mouth and tongue, dry, coarse, and brittle hair, spoon-shaped finger-nails (koilonychia) and menstrual disturbances.

Examination reveals pallor of the skin and mucous membranes. There is rarely any splenomegaly. Blood examination discloses a hypochromic microcytic anæmia with often a very low colour index (*e.g.*, 0.3 to 0.4 and Mean Corpuscular Hæmoglobin Concentration less than 30 per cent.). There is no evidence of hæmolysis. The bone marrow is normoblastic in type. Achylia or achlorhydria is commonly found.

Treatment.—Blood transfusions may be needed if the hæmoglobin is very low—say below 25 per cent. (3.7 G. per 100 ml.). Care should be taken to avoid circulatory overloading (*see page 48*).

Compound Ferrous Sulphate Tablets B.P.C. 0.3 to 0.6 G. and ascorbic acid 50 mg., three times a day, may be given additionally

but the most satisfactory and the quickest route in very ill patients is to give a saccharated iron preparation intravenously (Ferrivenin, Benger; Iviron, British Schering; Neo-ferrum, Crookes). The total dose is 37.5 mg. for each 1 per cent. (Haldane) deficit in hæmoglobin; 25 mg. should be given on the first day, 50 mg. on the second day and then 100, 150, and 200 mg. on each succeeding day and continuing thereafter with 200 mg. daily until the total calculated amount has been given. Neo-ferrum appears to be the least toxic and can be given in increasing doses up to 500 mg. at a time. It is important to make sure that the injection is entirely intravenous. Intramuscular iron preparations act too slowly to be of value in emergencies.

‘**Hæmolytic anæmias** all show the features common to any anæmia, together with a varying degree of jaundice. We are concerned here only with the acute types such as the hæmolytic anæmia of Lederer, hæmolytic crises in acholuric jaundice, erythroblastosis foetalis (*see page 298*), and hæmolytic anæmias from known causes (*e.g.*, gold, arsenic, benzene, etc.), malaria (*see pages 326 and 367*). Hodgkin’s disease, leukæmia, and the rare Marchiafava’s paroxysmal hæmoglobinæmia with hæmoglobinuria. Hæmolytic crises may be precipitated by severe infections which must be looked for and treated. Occasionally acholuric jaundice is complicated by biliary colic caused by pigment stones (*see page 80*).

Treatment.—Rest in bed and immediate removal of toxic hazards are clearly indicated. BAL (British Anti-lewisite. Dimer-caprol) should be given promptly (*see page 265*) where metallic poisons such as arsenic, gold, bismuth and mercury are concerned. Some still use sodium thiosulphate 0.4 to 0.6 G. in 10 per cent. solution intravenously for gold intoxications. Cortisone 200 to 300 mg. daily or prednisolone 30 to 50 mg. daily in prolonged courses of several weeks or months may be of great help in some cases.

Repeated blood transfusions are usually needed initially. Splenectomy is the best treatment for acholuric jaundice, but is only rarely needed as an emergency measure. In Marchiafava’s hæmolytic anæmia intensive alkalinisation of the urine is important. Potassium citrate or sodium bicarbonate in doses of gr. 60 should be given three-hourly.

POLYCYTHÆMIA

This disease usually presents in a patient over 45 with severe headaches and sometimes with painful flushing of the skin of the legs. The skin, especially of the face, is dark red or plum-coloured. There is a variable degree of splenomegaly. The diagnosis is therefore obvious before complications arise. Occasionally the presenting feature is cardiac failure, hyperpiesis, or even cerebral hæmorrhage or thrombosis. A few patients come with acute erythromelalgia of the lower limbs and some with symptoms of an acute abdominal crisis. Treatment is symptomatic and venesection may be the immediate method of choice for urgent cardiac and hypertensive symptoms. Where there are serious thromboses (*e.g.* cerebral) immediate anticoagulant therapy (page 595) must be started.

While achlorhydria is common in polycythæmia, a high gastric acidity is occasionally found. This may be associated with symptoms of acute duodenal ulceration, which may complicate the picture and call for prompt additional measures.

LEUKÆMIAS

Chronic myeloid and lymphatic leukæmias may present as emergencies not only because of weakness and dyspnœa but also on account of severe anæmia which may be hæmolytic in type. This may be very marked, particularly after X-ray or radio-active treatment. Acute abdominal pain may result from perisplenitis. Marked splenomegaly is practically a constant finding in chronic myeloid leukæmia and some degree of enlargement of the liver is common. Glandular enlargement is normally seen to a greater degree in chronic lymphatic leukæmia. Acute tonsillitis and acute ulceration of the skin, both due to localised leukæmic deposits, are not unusual features.

These clinical findings suggest the diagnosis which is confirmed by the high total white count (up to 600,000 or 800,000 per cu.mm.) with corresponding changes in the differential count according to the type. In myeloid leukæmias, myelocytes, promyelocytes and myeloblasts are found but, in lymphatic leukæmia, lymphocytes and lymphoblasts.

In acute leukæmias and monocytic leukæmia the problem is less easy but it is always an emergency. They are more commonly seen in children and young adults. The onset is often insidious and the

symptoms protean. When seen as acute conditions, they often complicate tooth extractions and similar emergencies or they follow sulphonamide therapy for pyrexial conditions which may, of course, have been the unrecognised beginnings of leukæmia. There is little or no splenic enlargement and, in the early stages, the peripheral blood picture is often more suggestive of pernicious anæmia, agranulocytosis or aplastic anæmia than a leukæmia. A sternal marrow biopsy will usually give the correct diagnosis, even in the early stages but *no time must be wasted* before beginning treatment.

Treatment.—Except in the terminal stages of chronic leukæmia, transfusion of whole blood and a first intravenous dose of 5 mg. of nitrogen mustard (Trillekamin, Crookes) will improve the patient sufficiently to enable specific anti-leukæmia therapy to be continued more actively. For acute leukæmia, transfusion is a temporary life-saving measure, followed immediately by intensive cortisone therapy (100 to 300 mg. daily for three weeks) or prednisolone 20 to 60 mg. daily for a similar time. While most of these acute leukæmias usually progress to fatal terminations in a few weeks, at least temporary remissions of several months and occasionally years can now be obtained in more than 30 per cent. of cases after intensive therapy. Some acute cases have had complete remissions of up to six years and chronic leukæmias for much longer.

ACUTE RETICULOSIS

Patients with acute reticulosis may present in severe relapses often resembling acute leukæmia or lymphosarcoma with enlargement of glands, spleen or liver. Rapid relief can very often be obtained by a packed cell transfusion (*see page 591*) and intravenous administration of nitrogen mustard (Trillekamin, Crookes) in doses of 5 to 6 mg., repeating as needed according to the white cell count. As in the treatment of acute leukæmias cortisone may be of great value. ,

AGRANULOCYTOSIS

This is one of two grave conditions calling for urgent treatment which should be thought of when a patient presents with a sore throat (the other being diphtheria). There may be also necrotic ulceration of the mouth, pharynx, rectum or vagina and a brawny swelling of the neck. The patient looks very ill. Generalised glandular enlargement and splenomegaly rarely occur.

The onset is fulminating and commonly follows the administration of certain drugs to which the patient is sensitive. These include sulphonamides, amidopyrine, thiouracil, epanutin-type drugs, gold, arsenic and bismuth salts given parenterally, radioactive agents and chloramphenicol.

In the circulating blood, the most characteristic finding is the virtual disappearance of granular white cells (counts of 300 or less per cu.mm. are typical) while the lymphocytes remain relatively normal in appearance and number. Erythrocyte and platelet counts and the hæmoglobin concentration are usually unaffected at first.

Treatment.—This is often disappointing and the outcome largely depends on prompt diagnosis and the elimination of the causative agent. Firm handling of the situation is imperative, from the moment the diagnosis is made, the patient and his attendants must be told explicitly that no medicines of any kind are to be given without the physician's consent. It must be explained that these include sleeping tablets and purgatives.

Pentnucleotide (Menley & James) (*see page 655*) 20 ml. can be given intramuscularly every four to six hours. But this is painful and early and repeated transfusions of fresh whole blood, containing 80 ml. of pentnucleotide to each 500 ml. seem to produce the best results.

Secondary infection of the necrotic lesions causes serious constitutional disturbances as well as the pyrexia. Penicillin or a similar antibiotic should be given intramuscularly and topically and mouth washes of saline and peroxide used. *Sulphonamides must not be given to these patients under any circumstances.* All other measures are of only secondary importance but claims have been made for leucocytic cream intramuscularly (injecting the buffy leucocyte layer obtained after centrifuging 150 ml. of blood), pyridoxine (Roche) 150 to 200 mg. intravenously, cortisone (200 to 300 mg. daily), prednisolone (20 to 60 mg. daily), and many other agents.

When the patient recovers he should be warned to avoid the toxic drugs already mentioned, even though they may not have been definitely incriminated as the cause of the illness.

HÆMORRHAGIC DISORDERS

While purpura occurs in thrombocytopenia, Henoch-Schönlein disease, hæmophilia, hypoprothrombinæmia, scurvy and other

bleeding conditions, it may be the first sign of acute leukæmia or aplastic anæmia. A clear understanding of the underlying defect is therefore essential for rational treatment depends entirely on the cause as determined by specific hæmatological examinations. Consequently, a patient with a bleeding diathesis should be referred as soon as possible to a hospital with adequate facilities for investigating blood disorders. Hæmorrhagic manifestations may be due to disorders associated with the platelets, acquired or congenital defects of the vessel endothelium, or disorders of blood coagulation.

DISORDERS OF PLATELETS

Thrombocytopenic purpura.

‘In this condition, a variable and sometimes severe degree of bleeding occurs in the form of multiple petechiæ and ecchymoses in the mucous membranes and skin, or there may be sudden painless hæmorrhages from the alimentary or renal tracts. Hæmorrhages into the brain and internal organs (*e.g.* the adrenals) occur in the severest cases. Secondary anæmia develops, and is proportional to the amount of blood lost. The significant finding is a much reduced platelet count (usually below 40,000 per cu.mm.). In addition, a prolonged bleeding time is found but the coagulation time is usually normal.

Thrombocytopenic purpura may be idiopathic or primary (as yet of unknown cause) or secondary to the use of many drugs such as gold salts, bismuth preparations, arsphenamine, many benzene derivatives, Sedormid, barbiturates, sulphonamides, thiouracil, snake venom and radio-active agents. It also occurs as a complication of infectious fevers, and in aplastic anæmia, acute leukæmia, agranulocytosis, hypersplenism and, sometimes, pernicious anæmia.

Treatment.—Since it may not be possible to decide in an emergency whether the condition is primary or secondary, any drugs which might have been responsible should be discontinued. Fresh whole blood of appropriate group and supplementary platelet suspensions should be transfused and repeated as necessary. The object is to give red cells and hæmoglobin to relieve the severe anæmia, and also to supply fresh normal platelets (which are not usually present in stored blood). Cortisone or A.C.T.H. have been recommended in these cases, but the results have been most disappointing. Nevertheless, they should be tried in the secondary type.

Splenectomy is contra-indicated in secondary thrombocytopenic purpura, but in the primary or idiopathic type it almost invariably cures the condition, and is definitely called for at the earliest opportunity. It is important not to wait for possible recovery from the attack, for the more ill the patient the more urgent is the need for splenectomy. It should be carried out after a preliminary transfusion of fresh whole blood, and in almost all cases the results are most satisfactory, the platelet count beginning to rise as soon as the splenic pedicle is tied. Splenectomy is important because of the risk of hæmorrhage into the brain and vital organs (especially the adrenals) which may occur without warning so long as the platelets remain low. After the operation the platelets rise steadily often to abnormally high levels, ultimately settling down to about 250,000 to 400,000 per cu.mm. In general, the prognosis is good for the primary group but very poor for secondary thrombocytopenic purpuras.

DISORDERS OF THE CAPILLARY ENDOTHELIUM

In these conditions purpura is associated with normal blood clotting properties and normal platelet function, the defect which may be acquired or congenital being in the capillary vessel wall.

Henoch-Schönlein purpura.

This is the main condition most commonly seen in this group. It may be allergic in character in some cases, but usually the ætiology is obscure. It is a type of non-hereditary, non-thrombocytopenic purpura in which the lesions in the skin and mucous membranes are associated with urticaria, joint swellings, abdominal pain and sometimes melæna. These latter symptoms may simulate a surgical emergency, and care is needed in diagnosis at this stage if there are hæmorrhagic manifestations and a history of previous attacks. Because of pains in the limbs it is sometimes called purpura rheumatica. The disease often begins in the second or third decade and continues throughout life. While it is rarely complicated by cerebral catastrophes as is thrombocytopenic purpura, it may cause considerable disability.

Treatment.—This is unsatisfactory. If the gastro-intestinal tract is involved, rest in bed and a light diet are indicated. For urticarial manifestations 0·5 to 1 ml. of Injection of Adrenaline B.P. subcutaneously, or ephedrine 16 mg. (gr. $\frac{1}{4}$) thrice daily by mouth (the last dose being given at 4 p.m.) may help, but usually one

of the newer anti-histamine drugs (*page* 611) is more valuable if given in adequate dosage orally or intramuscularly. When an allergic basis can be established by skin tests or by the history, desensitization, using the appropriate solutions (supplied by C. L. Bencard, Ltd., Minerva Road, Park Royal, London, N.W.10) should be tried. Aspirin should be given in the rheumatic type together with vitamin K, or its analogue (*page* 599). Splenectomy is of no value in this type of purpura. Appropriate anti-anæmic treatment may be necessary. More recently, disorders of the capillary walls as in Henoch-Schönlein purpura, and other conditions such as vascular pseudohæmophilia, and multiple telangiectasia have been treated with Adrenoxyl (Horlicks) (3 to 6 tablets of 2.5 mg. each daily by mouth, and Naphthionin (Maw) (10 ml. = 1 mg. intravenously). Their therapeutic value has yet to be proven. Severe bleeding from nasal telangiectasia can be so serious as to need more drastic treatment such as local cauterisation, packing with Calgitex, fibrin foam, spraying with a concentrated thrombin solution, and even blood transfusion (*see* Epistaxis, *page* 123).

DISORDERS OF BLOOD COAGULATION

Without considering the mechanism of blood coagulation in detail here it is clear that the hæmorrhagic manifestations that arise following defects in this system fall into three groups—(1) defects of thromboplastin generation, (2) disorders of the prothrombin complex, and (3) impaired thrombin-fibrinogen reaction.

DEFECTS IN THROMBOPLASTIN GENERATION

Hæmophilia and Christmas Disease.

Defects of thromboplastin generation may arise as a result of a congenital deficiency of the antihæmophilic globulin as in classical hæmophilia, or the Christmas factor in the so-called Christmas disease—a hæmophilia-like disease. A similar condition may be seen when circulating anticoagulants are present in the blood and interfering with the action of these two factors. The resulting clinical manifestations in these three conditions are identical and special laboratory tests are needed to differentiate them. The significant findings on blood examination are a prolonged coagulation time and abnormal prothrombin consumption.

Serious emergencies requiring very urgent treatment may arise suddenly in these cases for bleedings may occur from many places

either for no apparent reason, or more often following some minor injury or operation such as tooth extraction. Occasionally the presenting symptoms are caused by severe intra-abdominal or intestinal hæmorrhage or hæmatemesis, and are distinguished from those of appendicitis or duodenal perforation only with great difficulty. Cerebral hæmorrhages and other bleeding manifestations may also arise. The resulting anæmia is only slight following hæmorrhage into a joint, but it may be severe after hæmorrhage elsewhere.

Hæmophilia and Christmas disease are hereditary, being seen almost entirely in males but transmitted by females. Very occasionally female hæmophiliacs have been reported. Hence there is often a family history to aid diagnosis, and every known hæmophiliac in the country should now be in possession of the green National Hæmophilia Card containing essential details for emergencies (*see page 638*).

Treatment.—These patients should be admitted to hospital at once for treatment. In spite of many claims, there is no curative treatment but much can be done to relieve the emergencies that arise. The patient should rest in bed until hæmorrhage has ceased. When there is bleeding into a joint, this should be wrapped in cotton wool and immobilised on a light splint. Alternatively, an ice bag may be applied but other active measures such as aspiration of blood are definitely contra-indicated. The suggested value of hyaluronidase injections into joints is unconfirmed and not to be recommended without further experience.

Surface bleeding as from a wound or tooth socket demands local treatment, but local measures *must not increase the local tissue trauma* so that suturing and cauterisation must not be used; they are useless and dangerous. Clots should be removed and small gauze dressings soaked in 1 in 10,000 solution of Russell Viper Venom (Stypven, Burroughs Wellcome; Rusven, Boots) or concentrated thrombin solution applied firmly. In the case of a tooth socket, they should be held in place by a small dental splint previously prepared.

Among the newer hæmostatic agents now available, the most potent is thrombin (Maw), which will produce almost instantaneous coagulation and therefore must *never* be given intravenously. It may be given by mouth as Thromboral (Maw) (*page 88*) for bleedings from the stomach or duodenum; dusted on as a

powder; applied in solution or sprayed on the bleeding surface after swabbing away the blood and old clots immediately beforehand. Other new absorbable hæmostatic agents are calcium alginate gauze (Calgitex), fibrin foam and gelatine sponge (Allen and Hanburys, Ltd.), all of which can be applied directly to the bleeding areas or even packed into wounds and cavities. Their efficiency can be enhanced considerably by soaking them first in a thrombin solution.

Another method is to apply to the wound a 5 per cent. solution of sodium alginate followed by a 0.5 per cent. calcium chloride solution. This provides an immediate coagulum of calcium alginate which seals the bleeding points. Calgitex powder may be dusted on a wound with similar effects.

Severe or prolonged bleeding usually calls for whole blood transfusions until hæmostasis is obtained and anæmia is relieved.

In hæmophilia the clotting defect can be corrected to varying degree according to the amount of antihæmophilic globulin (AHG) given either in the form of fresh whole blood, fresh plasma or preparations made from ox or pig plasma. AHG cannot yet be stored in blood or plasma as potency is lost rapidly, so transfusion of fresh blood must be commenced within an hour or so of collection from the donor. It is known that blood transfusion equal to at least half of the total blood volume of the patient is required to bring the thromboplastin test to within the normal range, although in actual clinical practice much more than this may be required to produce satisfactory hæmostasis. Daily blood transfusions are usually required in the beginning of treatment and then very great care must be taken to preserve the veins for future needs.

In Christmas disease the clotting defect can be dealt with a little more easily for the deficient Christmas factor does not lose much potency on storage and so stored blood or plasma can be used as well as fresh blood, but AHG preparations are of no value, since AHG is not deficient in this disease.

Emergency operations always carry grave risks for hæmophiliacs and should be avoided if possible. They necessitate transfusions of fresh whole blood, preferably by the drip method before, during and after operation. Fresh plasma or antihæmophilic globulin may be given intravenously if available. Even when satisfactory hæmostasis has been achieved there may be considerable delay in healing due to the formation of faulty granulation

tissue. As a rule these patients are best referred to the special Hæmophilia Centres now set up in certain regions (*page 638*) to deal with such cases.

DISORDERS OF THE PROTHROMBIN COMPLEX

Many of these may be due to deficiencies of such substances as Factor V, Factor VII, prothrombin, etc., and manifest themselves as bleeding conditions resembling the diseases already discussed above; they cannot be differentiated without the aid of very special hæmatological tests and for this reason these patients must be referred as quickly as possible to appropriate centres for diagnosis and control of treatment.

Hypoprothrombinæmia.

This condition, which is due to a deficiency of vitamin K, may disclose itself as a purpuric or an hæmorrhagic condition. It most commonly arises from faulty intake or availability of vitamin K in the diet, poor absorption from the alimentary tract, or faulty liver function with inadequate conversion of vitamin K to prothrombin. Thus vitamin K₁ oxide or K₁ analogue (*page 599*) should be given to the infant with hypoprothrombinæmia (hæmorrhagic disease of the newborn) and some pregnant women may also need it as a prophylactic measure.

Severe hæmorrhagic features may be seen in association with obstructive jaundice and hepatic cirrhosis due to faulty absorption or poor conversion of the vitamin K to prothrombin. Treatment is best given in the form of vitamin K₁ or K₁ analogue (*page 599*) by injection as oral forms are not absorbed in these cases.

Perhaps the most serious hæmorrhagic disorder seen in this group is that due to overdosage with the anticoagulant drugs, although this usually arises when they have been given without proper laboratory control.

Treatment in these cases is very urgent. The anticoagulant drug must be stopped at once and vitamin K₁ or K₁ analogue (*page 599*) given, repeating in four hours if the prothrombin time has not returned to almost normal. Fresh whole blood transfusions will also be required. Transfusions of serum (which contains Factor VII) may also be of value, perhaps alternating with whole blood.

DISORDERS OF THE THROMBIN-FIBRINOGEN REACTION

Severe hæmorrhages may be seen in patients with congenital or acquired fibrinogenopenia, or having heparin-like anticoagulants* in the circulating blood. These are rare conditions and the former condition is best treated by giving freeze-dried human fibrinogen obtainable from the Medical Research Council Blood Products Unit at the Lister Institute of Preventive Medicine, Dagger Lane, Elstree, Herts (Tel. Elstree 1009). This powder is dissolved in the indicated amount of sterile saline and given slowly intravenously. *Further amounts should be given until hæmostasis is produced.

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CHAPTER X

Fits, Faints and Unconsciousness

IT is intended here to give general directions only on how to deal with those conditions in which the urgent call is occasioned by a sudden illness in which consciousness is clouded or lost.

These cases fall into two groups:—

- (1) Those in which the “fit” or “faint” is of short duration—
“episodic unconsciousness” due in most cases to epilepsy, hysteria or vaso-depressor syncope.
- (2) Those in which unconsciousness is profound (*i.e.*, coma) and relatively prolonged.

FITS AND FAINTS

Although the dividing line between fits and faints is less sharp than was formerly believed it is still convenient to consider them separately. Almost always the attack is over or the patient obviously improving by the time the doctor arrives. Three questions should arise in his mind when called to such a case.

- (1) Is it a fit (*i.e.*, neurological in origin)?
- (2) Is it a faint (*i.e.*, cardio-vascular in origin)?
- (3) Is it some other kind of attack?

Is it a fit?

This question may be easy to answer if there is a history of previous attacks competently observed. The occurrence of clonic movements suggests that the attack is a fit or epileptiform attack. Incontinence of urine, tongue biting, and injury in the attack also point strongly, though not conclusively, to epilepsy (but do not indicate its cause). Occasionally a daytime fit may bring the doctor to a patient whose previous nocturnal attacks were unrecognised. A history of waking with a headache and a bruised feeling in the limbs suggests convulsions during the night. Unequivocal evidence of organic nervous disease, such as cerebral tumour or general paralysis of the insane, suggests that the attack was a fit.

Minor epilepsy or petit mal as a cause of "fainting" may be hard to diagnose since there are no clonic movements. Loss and recovery of consciousness are sudden, producing a mere hiatus in cerebration; post-epileptic phenomena (automatism, etc.) have special diagnostic value but they are relatively rare. In a cardiovascular "faint" consciousness is more gradually lost and regained. It is generally preceded by dimness of vision since the retina is specially sensitive to oxygen lack. Scars on the tongue and congenital epidermal defects suggest epilepsy.

Is it a faint?

The numerous conditions of cardiovascular origin which come under this heading may be placed in two groups.

- (1) Where there is clear evidence of heart disease, *e.g.*, heart block, aortic incompetence, etc.
- (2) Where there are no signs of cardio-vascular disease, *e.g.*, vaso-vagal attacks, postural syncope, carotid sinus syncope, and transient heart block.

Is it some other kind of attack?

Two common ones should be considered.

(1) Hypoglycæmia. A clear history of insulin injected and not followed soon enough by a meal will usually point to the diagnosis but difficulties will arise if no witnesses are present. Search should be made for needle marks and a diabetic card or literature in the pockets. If in doubt, dextrose should be injected intravenously (*see page 245*). Hypoglycæmia may, rarely, be spontaneous.

(2) Internal hæmorrhage—as from a duodenal ulcer. There may be a history of dyspepsia. Increasing pallor, increasing rapidity of pulse, dyspnoea and restlessness should suggest that hæmorrhage is the cause of the faint.

COMA

Here unconsciousness is more prolonged than in a "fit" or a "faint." A classified list of causes of coma, while useful for detailed discussion, is not so valuable when faced with a comatose patient as is a simple plan of enquiry. This is therefore given first.

(1) Is there a previous history of disease which might cause coma?

e.g.

Diabetes (test the urine for ketone bodies [*see page 232*] and sugar).

Insulin Coma. (If this seems likely give 20 ml. of 60 per cent. dextrose [= 12 G.] intravenously).

Nephritis (test the urine for albumin).

Perilious malaria (examine thick and thin blood films). In persons reaching England by air from the tropics the first attack may occur here.

(2) Does the immediate history point to the cause?

e.g.

Injury. (Do not too readily assume it is the whole cause).

Poisoning (including alcohol).

Epilepsy. (Fits may be symptomatic of other disease, *e.g.*, meningitis).

(3) Are there any physical signs of disease of the central nervous system?

e.g.

Cerebral vascular accidents. (Lumbar puncture may be necessary).

Meningitis. (Lumbar puncture is essential).

To answer these questions a full examination will have to be made and the findings recorded. This should always include the urine, the pupils and fundi, the heart rate, the blood pressure and the temperature.

CAUSES OF COMA

(1) Injuries. (Concussion [*page 195*], Cerebral compression [*page 195*], Electric shock [*page 118*]).

(2) Increased intracranial tension. (Cerebral tumour [*page 198*]).

(3) Cerebral vascular accidents. (Hæmorrhage, thrombosis, embolism [*page 207*]).

(4) Meningitis (*page 200*). Encephalitis (*page 198*). Cerebral malaria (*pages 326 and 368*).

(5) Effects of heat (*page 338*).

(6) Exogenous poisons (*page 4*). (Alcohol, barbiturates, coal gas, etc.).

- (7) Endogenous poisons. (Uræmia [*page* 269], diabetic coma [*page* 238], cholæmia [*page* 81]).
- (8) Hypoglycæmia (*page* 242).
- (9) Epilepsy (*page* 198).
- (10) Circulatory disturbances. (Syncope [*page* 150], Stokes-Adams attacks [*page* 151]).
- (11) Hysteria (*page* 203). Schizophrenia.

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CHAPTER XI

Neurological Emergencies

THE chief neurological conditions which constitute emergencies are unconsciousness, convulsions, sudden paralysis, sudden blindness, severe pain and vertigo. These symptoms are not mutually exclusive, but one is usually predominant. In this chapter the causes of each symptom are outlined and the underlying diseases are discussed, but only *immediate* treatment is described.

UNCONSCIOUSNESS

The general problem presented by the comatose patient should be approached on the lines suggested in Chapter X.

Although the mechanism of all types of coma depends ultimately on damage to the nervous system, we deal in the following sections only with the essentially neurological causes of coma.

MANAGEMENT OF COMA OF NEUROLOGICAL ORIGIN.—Most patients unconscious from neurological causes have to be removed to hospital. Exceptions are those who have attacks of short duration, such as vasomotor attacks, epilepsy, and *proved* hysteria. It is sometimes wisest to keep a patient with spontaneous subarachnoid hæmorrhage at home, at any rate at first, lest his condition deteriorate under the stress of transportation. Pending a diagnosis one must not overlook the needs of the unconscious patient (*i.e.* catheterisation, nasal feeding and avoidance of bed sores).

These immediate points having been covered, attention can be given to the middle-distance policy, where the chief risk is pneumonia. Aspiration of mucus from the throat should be performed hourly if necessary. Injection of Nikethamide 2 to 4 ml. intramuscularly every two to four hours may aid a failing circulation. Prophylactic penicillin should be given (*see page 601*).

Once general causes have been excluded, examination of the nervous system should be as full as possible but may have to be modified because of the state of consciousness. Certain special devices in addition to the routine examination may be helpful in diagnosis and localisation of the lesion.

1. Can the patient be roused? (*i.e.*, Is it deep sleep rather than coma?).
2. Note the state of the corneal reflexes and pupillary responses and whether the head and eyes are deviated to one side. (In cerebral hæmorrhage they tend to be deviated towards the side of the lesion).
3. Apply painful stimuli and see if there is absence of movement in one or more of the limbs, or the face. (The patient may show signs of feeling pain and yet be unable to move).
4. Test the grasp reflex by drawing two fingers across the patient's palm. If positive, the patient closes his hand and grasps the stimulating fingers firmly though involuntarily. When present it may indicate a lesion in the contra-lateral frontal lobe.
5. Kernig's sign (attempts to extend the flexed knee on the flexed hip are prevented by spasm of the hamstrings) and Brudzinski's sign (flexion of the knee and hip produces involuntary flexion of the opposite knee and hip) will indicate the presence of meningeal irritation.
6. In cases of injury, comparative examination every two or three hours may be necessary to gauge progress and to discover localising signs.

THE CEREBRAL VASCULAR DISTURBANCES

"Sudden spontaneous happenings within the cranium are vascular in origin." Vascular accidents may be caused by:—

- (a) thrombosis and embolism (*page 207*).
- (b) hypertensive attacks (*page 267*).
- (c) extra and sub-dural hæmorrhage (*page 198*).
- (d) vasospasm (*page 209*).
- (e) **spontaneous subarachnoid hæmorrhage.** This may occur at any age and result from aneurysms (congenital and acquired), tumours and blood diseases. Sometimes it is associated with coarctation of the aorta or bacterial endocarditis. The catastrophe, which may be unheralded, or preceded by cranial nerve palsies and headache, is ushered in with sudden intense headache, and vomiting. These symptoms and the accompanying signs, arising from a combination of meningeal irritation and increased intra-cranial pressure, tend to vary with the degree of hæmorrhage.

On examination, apart from coma, signs of meningeal irritation are discovered. The plantar reflexes are often extensor; papilloedema with or without retinal hæmorrhages may be seen. Pyrexia and albuminuria may be present. The final diagnosis depends on the examination of the C.S.F. which is found to be uniformly blood-stained in successive samples, and indeed may be so heavily contaminated as to raise doubts as to whether the theca or a vein has been punctured (*see page 525*).

Treatment.—The problem is to steer the patient between the Scylla of increased intracranial tension and the Charybdis of prolongation or recurrence of bleeding. The patient must be confined to bed in all cases (usually for four to six weeks). If the C.S.F. pressure is high (over 250 mm.) at the time of the first lumbar puncture it should certainly be lowered, but not more than 15 ml. of fluid should be removed. Further withdrawal is necessary if the blood pressure rises or other signs of cerebral compression, such as deepening coma, absence of corneal reflexes, and fixed pupils, appear.

Headache should be treated by analgesics, and a good combination containing heroin (diamorphine), *if available*, is given on *page 221*. Opium derivatives should not be used on account of their depressant effect (often fatal) on the respiratory centre. If a cardiac valvular lesion is detected arrange for blood culture and sensitivity tests to be made and begin penicillin treatment for bacterial endocarditis. If the patient's condition deteriorates emergency neuro-surgical intervention may be needed to attempt to arrest the bleeding, particularly if the lesion is an angioma. Each case must be judged on its merits since the operation is hazardous and in any case must depend on demonstration of the site of bleeding by cerebral angiography. When bleeding continues it is possible to make a broad differentiation between the young patient with a ruptured congenital aneurysm and the elderly patient whose bleeding is due to arterio-sclerotic changes. In the former immediate angiography with direct operation on any aneurysm shown is probably the correct course whilst in the latter it is wiser to use conservative measures at the time of the hæmorrhage and then to consider tying the internal or common carotid artery in the light of the later angiographic evidence.

(f) **Intracerebral hæmorrhage.**

This is most commonly caused by a rupture of an atheromatous artery in a patient who has hypertension, the commonest sites being in the internal capsule (Charcot's artery) and the pons. There is often a previous history of headache and of visual, motor and speech disturbances. When hæmorrhage occurs, the patient becomes dazed and then comatose, or may have convulsions.

Unconsciousness, and stertorous breathing, often of Cheyne-Stokes character, are common in all types, the other findings varying with the site of the hæmorrhage.

(i) **INTRACAPSULAR HÆMORRHAGE.**—The whole capsule is usually involved with correspondingly widespread effects. The head and eyes are turned to the side of the lesion as a result of paralysis of the connections for rotation of the head to the opposite side. Paralysis of the face, arm, and leg may be demonstrated in the comatose patient by observing the response to painful stimuli and by the *absolute* flaccidity as compared with the normal side.

The tendon reflexes are commonly lost in the paralysed limbs at first but an extensor plantar response is usually present, and may be bilateral. Hemi-analgesia may be demonstrable.

(ii) **PONTINE HÆMORRHAGE.**—Because of the concentration of nerve tracts in the pons, damage here also produces widespread effects. Early signs may be crossed—showing facial paralysis on the affected side, and flaccid paralysis of the opposite limbs, but the effects soon become bilateral.

Characteristically, the pupils are of "pin point" size. The heat regulating mechanism may be paralysed and, since the body is then poikilothermic, injudicious application of excessive warmth results in hyperthermia.

(iii) **INTRA-VENTRICULAR HÆMORRHAGE.**—This usually complicates intra-capsular bleeding. Coma is deep and is associated with bilateral signs, meningeal irritation, and often terminal hyperthermia.

Treatment.—This is on the lines suggested for subarachnoid hæmorrhage. Surgical intervention may be required either to aspirate intra-cerebral clot or to attempt to arrest the hæmorrhage.

Prognosis.—The relatives will put urgent questions as to prognosis and should be warned of the uniformly grave outlook. Grave prognostic signs in the early stages are:—

- (a) Coma lasting 48 hours or longer, or progressively deepening.
- (b) Undue fall in blood pressure after the initial rise.
- (c) Rise in temperature, often to extremely high levels, e.g., 106° F.
- (d) Rising pulse and respiratory rate.
- (e) Evidence that the bleeding is intra-ventricular, or into the pons.

(g) **Cerebral venous sinus thrombosis.**

This is discussed here since it usually causes disturbance of consciousness, although convulsions may also occur. It is customary to describe sinus thrombosis as *primary* when it occurs at the extremes of life (usually in association with debilitating illness), or when it is part of the picture of thrombophlebitis migrans, and *secondary* when it complicates injury or infection of the skin, subcutaneous tissues and bones of the head and face, or the air sinuses. It may also be secondary to remote infections, and particularly puerperal sepsis or pelvic thrombosis.

Headache, vomiting, delirium, and swinging temperatures with rigors may occur in all types. In addition, special symptoms and signs are found, depending upon the sinus involved.

(i) **CAVERNOUS SINUS THROMBOSIS.**—Pain is localised in the forehead and the affected side of the head. There is œdema of the eyelids and unilateral exophthalmos. The second, third, fourth, fifth and sixth cranial nerves may all be involved.

(ii) **SUPERIOR LONGITUDINAL SINUS THROMBOSIS.**—This is commoner in infancy, and frequently causes convulsions. There is head retraction and congestion of the scalp veins. Signs of bilateral pyramidal tract involvement may be found in the legs.

(iii) **LATERAL SINUS THROMBOSIS.**—This causes pain round the ear and venous congestion over the mastoid process. Slight involvement of pyramidal tract function is sometimes seen.

Treatment.—Surgical intervention may be necessary if a suppurating focus is present. In lateral sinus thrombosis, ligation of the internal jugular vein may be needed.

In all infective cases as basic treatment, penicillin (*page 601*) in conjunction with sulphonamides (*pages 599 and 608*) and anti-coagulant therapy (*page 595*) must be used. Other antibiotics may be needed (*page 605*).

THE MEDICAL ASPECTS OF HEAD INJURIES

The fact that unconsciousness is the result of injury is usually obvious, since even if witnesses are absent there are signs of trauma. The clinical state may be due to concussion, cerebral contusion or cerebral compression.



FIG. 19

Hutchinson's pupils: second stage.

Opposite side to lesion: Normal. Side of lesion: Dilated; reacts to light.

(Hamilton Bailey: *Physical signs in Clinical Surgery*)

Stage	Pupil on opposite side to the lesion		Pupil on side on which compression commenced	
1	Normal.		Slightly contracted. Sluggishly reacts to light.	
2	Normal		Moderately dilated. Reacts to light.	
3	Moderately dilated. Reacts to light		Widely dilated. Does not react to light.	
4	Widely dilated Insensitive		Widely dilated. Insensitive.	

FIG. 20.—The four stages of Hutchinson's pupils.

The first stage is rarely seen. It is the second (Fig. 20) and third stages that are of signal diagnostic importance. These changes are produced by initial irritation and subsequent paralysis of the third nerve, usually by subtentorial herniation of the hippocampal gyrus.

Concussion.

A blow on the head may produce all grades of altered consciousness from dazedness to coma. Such signs as occur in addition to unconsciousness are usually caused by impairment of function of the brain stem. Thus the pupils are dilated and inactive, the tendon reflexes are lost, the skin is pale and the blood pressure low. As the effect of the blow subsides, a return of function is observed, beginning with visceral activity, particularly vomiting. Recovery of consciousness takes place in stages, and patients often pass through a stage of automatism or one of delirium and irritability. Headache is invariable. In uncomplicated cases the symptoms clear up in 48 to 72 hours although headache may last longer.

Contusion.

The patient has usually been concussed but does not recover in the usual way. His condition deteriorates and death may follow. In less severe cases, consciousness returns, but somnolence alternates with great restlessness—the so-called cerebral irritation in which “the mechanism of the body is intact but the government has departed.” Focal signs may develop according to the area involved.

Cerebral compression.

This is caused either by extradural hæmorrhage (from the middle meningeal artery) or subdural hæmorrhage (from rupture of the brain or venous sinuses). It is characterised by *deepening* of unconsciousness and loss of corneal reflexes. It may not develop until some time after the injury and this *progressive* coma following a *lucid interval* is especially significant. Careful observation of the pupils may aid in diagnosis since they go through a series of changes as shown in Figs. 19 and 20 (Hutchinson's pupils). In addition, watch should be kept for such focal signs as may develop to indicate the side of the primary compression, though false localising signs are very common. The reader's attention is drawn to Fig. 21, which shows the clinical possibilities in diagrammatic form.

MANAGEMENT OF HEAD INJURIES.—Almost all closed head injuries are medical emergencies in the first place though surgical intervention may be required for complications. All patients who have lost consciousness for more than a few seconds should be

admitted to hospital. After taking the history and making a physical examination (which must include the scalp) the skull should be X-rayed. A period of observation should then follow during which the march of events is noted. Lumbar puncture and pressure reading should be delayed until the initial shock passes off, but successive punctures may be necessary to detect rising pressure. In the presence of papilloedema lumbar puncture is unnecessary and indeed, contraindicated.

Repeated examination of a patient unconscious as a result of head injury is necessary in order to detect evidence of brain compression. In addition to assessment of the neurological status, instructions should be given for the recording of pulse and respiration rates at half-hourly intervals with blood pressure readings every hour. Slowing of the pulse with elevation of the blood pressure may be the first valuable clues to increasing intracranial tension. Meantime the general principles of management of the unconscious patient should be applied with special attention to the maintenance of a free airway and the avoidance of aspiration. The patient should be placed on his side and if cyanosis is present oxygen should be given. If the unconsciousness is prolonged beyond 24 hours it is as well to combat the risk of infection both locally and in the chest by the administration of penicillin and sulphadiazine. Further, in such cases the patient should be turned every four hours, fluid intake must be maintained, and footdrop should be avoided by appropriate splinting.

CONCUSSION.—As consciousness returns, sedatives should be given. During the first 24 hours, attention must be paid to the level of consciousness, pulse rate, and the development of any neurological signs. If the first day passes without complications a decision must be made as to the proposed length of stay in bed. The practitioner should sum up the case not only from the standpoint of the present crisis but also from that of the patient's general, social and psychological background.

The length of post-traumatic amnesia should be assessed since its duration is a rough, though not infallible, guide to prognosis. Thus, mild cases of post traumatic amnesia of less than one hour should be up in three to six days, active in 10 to 14 days, and at full work within a month. In the early stages, the patient should be nursed in the position of greatest ease, and need not suffer the discomfort of being nursed flat. Gradual resumption

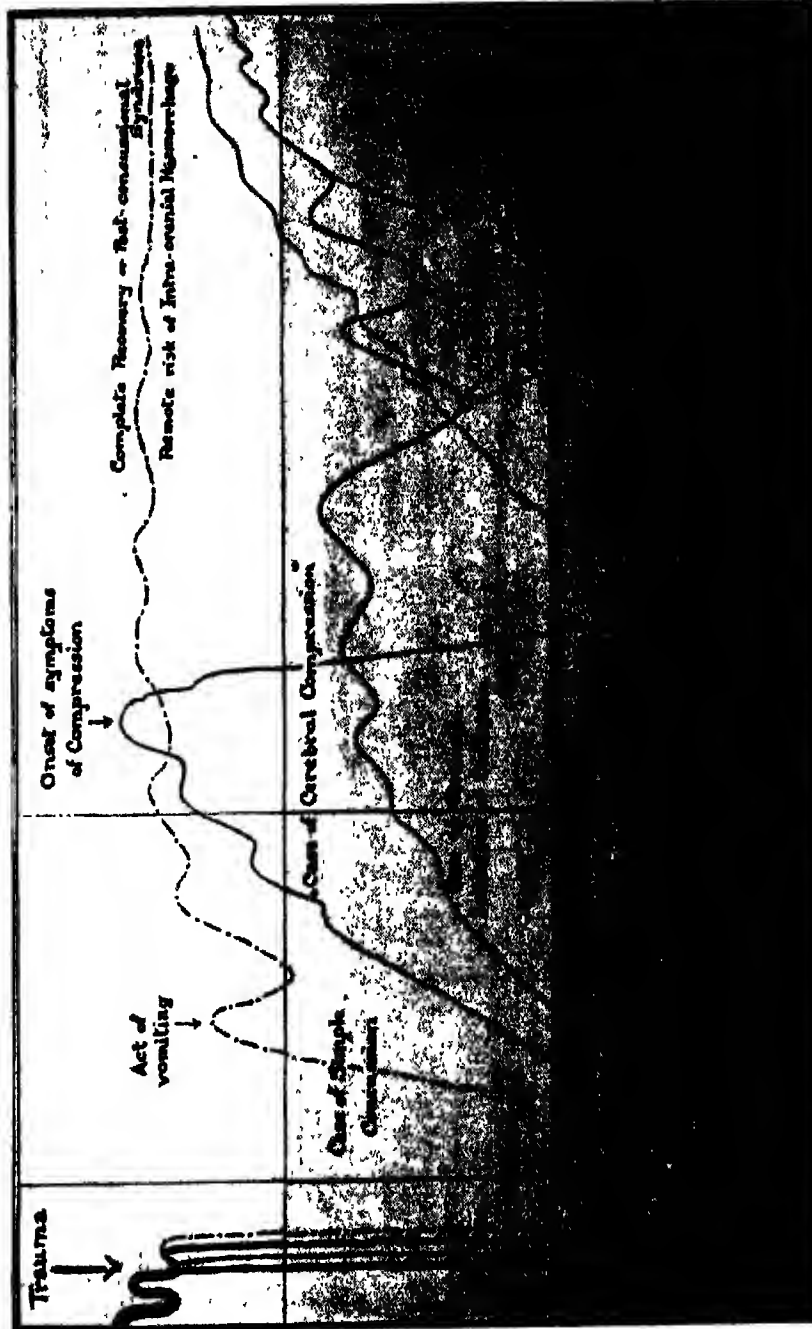


FIG. 21—The effects on consciousness of a head injury.

Clinical Type

- The case of simple concussion.
- The case of cerebral irritation.
- The case of cerebral compression.
- The case of cerebral laceration.

Pathological Basis

- No anatomical lesion. Probably a physiological change.
- Generalised cerebral oedema or cerebral contusion.
- By bone; by foreign body; by hæmorrhage; (a) extradural; (b) sub-dural—(1) diffuse, (2) localised.
- Actual laceration of cerebral tissue.

of mental and physical activities then follows, care being taken to be neither over-solicitous nor over-enthusiastic in spurring the patient on.

CONTUSION.—From the point of view of treatment, this may be regarded as an exaggerated form of concussion. Some patients benefit by restriction of fluid intake to 850 ml. (30 fl. oz.) daily. Magnesium sulphate enemata every six hours may help to reduce intra-cerebral pressure. 170 ml. (6 fl. oz.) of a 50 per cent. solution should be injected slowly into the rectum.

Six heaped tablespoonfuls (90 G. approx.) of Epsom Salts and 9 tablespoonfuls (130 ml. approx.) of water yield approximately 6 fl. oz. (180 ml.) of a 50 per cent. solution. Alternatively a vessel if half filled with crystals of Epsom Salts will yield a 50 per cent. solution when the crystals are dissolved and the vessel topped up with water.

Only if such measures fail should reduction of intracranial pressure by lumbar puncture be used.

COMPRESSION.—Surgical assistance is often required to deal with compound fractures, but apart from this, neurosurgical aid may be called for if the period of observation leads one to the conclusion that progressive compression is taking place.

CEREBRAL TUMOUR

This is usually diagnosed long before any disturbance of consciousness occurs. Occasionally, however, hæmorrhage or oedema in a tumour may cause sudden coma. The patient may then be tided over the immediate emergency by the judicious use of hypertonic (50 per cent.) sucrose—up to 50 ml. by slow intravenous injection, or magnesium sulphate enemata (*see above*).

EPILEPSY

Sometimes it is known that an unconscious patient is an epileptic. If fits have been frequent, the doctor is likely to be summoned urgently only if unconsciousness has been unduly prolonged, and in such circumstances the possibility of barbiturate overdosage as a cause of coma in a known epileptic should be remembered.

ENCEPHALITIS

The clinical picture of headache, pyrexia and disturbance of consciousness, with or without focal signs, may be produced by any of the types of encephalitis of which the following is a simple classification:—

Suppurative encephalitis (brain abscess).

- Non-suppurative encephalitis. (i) Complicating fevers, measles etc. (*page 317*).
(ii) Resulting from poisons—arsenic (*page 354*), organic solvents (*page 351*), and botulism (*page 72*).
(iii) As a primary affection of the brain.

Suppurative encephalitis.

This may present as a medical emergency when it complicates sepsis elsewhere in the body such as middle ear disease, sinusitis, pulmonary suppuration and pyæmia.

In the acute stage extensive involvement of brain tissue may occur with diffuse cerebral softening. Sometimes extension to the ventricle occurs early, causing virulent meningitis. When less virulent, a localised abscess forms and the clinical picture is similar to that of cerebral tumour unless and until rupture into the subarachnoid space occurs. The abscess may be extra-dural, sub-dural, intra-cerebral or intra-cerebellar so that the localising signs vary. The extra-dural and sub-dural abscesses tend to be complicated by venous sinus thrombosis.

Treatment.—Acute diffuse suppuration and rupture of a local abscess were, before the advent of penicillin, intractable conditions with an almost hopeless prognosis. Systemic administration of penicillin (*see page 601*) and for the first five days, intra-theal penicillin (25,000 units in 5 ml. of water diluted with further 5 ml. of C.S.F.) may offer hope of recovery, and should be combined with full sulphonamide dosage (*see page 599 and also page 608*). Should the organism be resistant to penicillin other appropriate antibiotics may be used. When an abscess forms, surgical intervention is necessary. It may be mentioned that electroencephalography is a most valuable investigation for the demonstration of the existence and site of an abscess, and it should be carried out at the earliest opportunity whenever this diagnosis is considered.

Virus encephalitis.

All types of virus encephalitis tend to occur in epidemics, and are of world-wide distribution. Sporadic cases also occur. They show no apparent seasonable preference for the colder months of the year, and appear to have no especially contagious characters.

SYMPTOMS.—The clinical picture varies in a most bewildering fashion. The principal acute symptoms, which may be sudden or gradual in onset, are headache, disturbance of consciousness, and affection of the cranial nerves. There may be inversion of the sleep rhythm with excitement, whilst pyramidal and extra-pyramidal manifestations with or without aphasia are common. Tendon reflexes vary but tend to be diminished. Involuntary movements of all types, including hiccup, may occur. Papilloedema has been noted. The C.S.F. shows slight excess of lymphocytes and a variable increase in the protein content.

Treatment.—In the absence of specific treatment, symptomatic measures are indicated. However mild the symptoms, the patient should be confined to bed. Hyperexcitability demands the use of sedatives, *e.g.*, hyoscine hydrobromide 0.32 to 0.65 mg. (gr. $\frac{1}{200}$ to $\frac{1}{100}$) six-hourly for two days, supplemented, if necessary, by phenobarbitone 0.1 G. (gr. $1\frac{1}{2}$) or more. If comatose, the patient should be dealt with on the general lines already indicated.

For encephalitis complicating infectious fevers *see page 317.*

MENINGITIS

All types of meningitis present a number of symptoms and signs in common. Severe headache is usual but the seriousness of the illness is more often proclaimed by clouding of consciousness. Meningitis is therefore considered here under the heading of coma. Diagnosis is rarely difficult; headache increases in severity and in severe paroxysms it may radiate to the neck and cause the patient to scream in anguish. The "meningitic syndrome" completes the picture; the victim lies curled up, resentful of interference, and showing photophobia. In children especially, vomiting and convulsions punctuate the progressive deterioration in the general condition. Pneumococcal meningitis can develop with surprising rapidity and by the explosive manner of its onset may simulate a cerebro-vascular accident.

Physical examination confirms the diagnosis and the signs can be explained on the basis of increased cerebro-spinal fluid pressure (causing retinal congestion and choked optic discs) and meningeal irritation (causing neck rigidity and the classical signs of Kernig and Brudzinski) (*see page 190*). If unrelieved, the increased intracranial pressure is inevitably reflected in depression of vital functions at various levels; slow and sighing respiration and inter-

ference with sphincter control are heralds of impending death. Pupillary abnormalities and disturbances of superficial reflexes are rarely absent but vary considerably from patient to patient, and in the same subject from hour to hour.

Given this combination of symptoms and signs the diagnosis of leptomeningitis is almost inescapable, but we must remember that the clinical picture may be caused by other pathological processes. These are:—

1. *Acute general infections* such as pneumonia which may produce headache and some of the signs (including an extensor plantar reflex) but not the serological evidence of meningitis (Meningism).
2. *Encephalitis* which may cause signs of meningeal irritation.
3. *Subarachnoid hæmorrhage* which closely simulates meningitis and in young persons with normal arterial systems may be clinically indistinguishable from it.

THE PRECISE DIAGNOSIS.—The diagnosis of leptomeningitis as opposed to the three conditions listed above, and the determination of the underlying cause, can be achieved only by examination of the cerebro-spinal fluid. The common organisms causing meningitis are the meningococcus, the pyogenic organisms such as streptococci, staphylococci and pneumococci, the tubercle bacillus, *Hæmophilus influenzae*, the virus of lymphocytic chorio-meningitis and other viruses and the spirochæta pallida.

Nevertheless, certain clinical findings may point to the probable diagnosis. Thus, meningococcal infection is suggested by a petechial rash and the occurrence of other cases, and also on rare occasions by severe collapse and shock (Waterhouse-Friderichsen syndrome, page 256). A pre-existing focus of infection such as otitis or pneumonia suggests a pyogenic meningitis. Fundal hæmorrhages may occur in subarachnoid hæmorrhage. An insidious onset, and a history of contact with an open case of tuberculosis is commonly found in tuberculous meningitis.

Meningitis is a rare complication of secondary syphilis. Its onset may be insidious with symptoms very similar to those of the meningo-vascular type of tertiary neurosyphilis. Occasionally, the picture is that of an acute meningo-encephalitis attended by convulsions, coma, and the signs of an acute meningitis. A similar picture may also occur as an episode

in the tertiary stage. As in the other types of meningitis, the diagnosis is established by the examination of the cerebrospinal fluid, which shows a lymphocytosis up to 1,000 cells per cu.mm. and a positive Wassermann reaction.

INTERPRETATION OF CEREBROSPINAL FLUID FINDINGS.—Inspection may reveal opalescence or turbidity thus establishing the existence of a pyogenic process, although in very early stages this may not be apparent to the naked eye. Specimens of fluid totalling 8 ml. should be sent at once to the laboratory for estimation of proteins, sugar, chlorides, cell count, Wassermann reaction, Lange curve, and above all, culture for the identification of the organism. If facilities for immediate laboratory examination are not available, begin treatment on the assumption that a turbid C.S.F. is due to pneumococcal meningitis not only because of the frequency of its occurrence but because this form of meningitis calls for early energetic treatment if the patient is to be saved. Any modification can be made later when the exact cause is known. If possible do the cell count and bacterial stain yourself immediately on one specimen and store a second specimen in the refrigerator for fuller investigation.

Treatment.

MENINGOCOCCAL MENINGITIS. (page 312).

PNEUMOCOCCAL, STREPTOCOCCAL, AND STAPHYLOCOCCAL MENINGITIS. (page 313).

Haemophilus Influenzae Meningitis. (page 313).

ACUTE LYMPHOCYTIC CHORO-MENINGITIS.—This disease is self limited and clears up in about fourteen days. No special therapy is required. The prognosis is excellent.

TUBERCULOUS MENINGITIS. (page 313).

SYPHILITIC MENINGITIS.—This "meningitis acutissima" has previously been treated by various intensive courses of arsenic and bismuth with varying success. Its relative rarity makes therapeutic trials necessarily restricted. *A priori* evidence, however, suggests that acute syphilitic meningitis should be treated by a full course of intra-theccal and intramuscular penicillin, as outlined for pneumococcal meningitis but continuing the intramuscular course for 21 days.

CONGESTIVE ATTACKS OF G.P.I.

These resemble cerebral hæmorrhage in their acute onset with interruption of consciousness varying from drowsiness and confusion to complete coma. No special emergency treatment is indicated.

HEAT PYREXIA ("HEATSTROKE") (*page 339*)

ELECTRIC SHOCK (*page 118*)

HYSTERICAL "UNCONSCIOUSNESS"

True unconsciousness does not result from hysterical causes. If a patient is genuinely comatose, organic disease must be assumed to be present even if there is a history of hysterical manifestations.

If hysteria is suspected an endeavour should be made to establish a definite diagnosis by showing a source of conflict. A full physical examination should be made. Normal ocular and tendon reflexes are strongly suggestive that there is no underlying organic disease. So also is the absence of stertorous breathing. If there is paralysis it is usually bizarre and any convulsive movements are irregular and do not follow the tonic-clonic succession of epilepsy. Flickering movements of the eyelids are commonly seen in these cases. In hysterical pseudo-coma, the doctor's attempts to open the eyes are resisted and pressure on the supra-orbital nerves or temporary obstruction of the nose and mouth will rapidly rouse the patient.

CONVULSIONS

Convulsions always present a serious problem in diagnosis and treatment. The occurrence of a "fit," especially for the first time, strikes terror into the mind of the layman, impels the immediate attendance of the doctor, and may betoken the onset of a serious disease requiring urgent treatment.

The functional unit consisting of upper motor neurone, lower motor neurone, and muscle, can react to insult in only two ways. It may become paralysed, or it may produce an abnormal contraction of the muscle. A repetition of muscular contractions, attributable to irritation of the upper motor neurones, constitutes the "convulsion" of clinical medicine. It is not surprising,

therefore, to find an imposing list of causes of convulsions and it is helpful from the point of view of diagnosis, to group these causes according to the age at which they occur.

Convulsions in children are dealt with on *pages 285 and 301*. Here we shall deal with convulsions in adults. The immediate problem may be to deal with the attack. If seen early enough, *i.e.*, before the jaws are in spasm, dentures should be removed and a handkerchief or soft gag should be inserted between the jaws. Spectacles should be removed and the collar loosened. Beyond preventing the patient from hurting himself, no restraint should be imposed. If no underlying disease is suspected, the patient may resume normal activities after 15 to 30 minutes' rest, but many will sleep for a long period and should be allowed to do so.

If the whole attack is witnessed, special points to observe are the origin and spread of movements; the development, if any, of unconsciousness, and the occurrence of incontinence, tongue biting and other injury. More often the attack is not observed and we have to rely on the evidence of a witness who is usually unreliable and often obtuse.

Enquiries and examination should be directed to settling the following points:—

- (1) Is the attack of psychological or organic origin?
- (2) If organic, is it attributable to neurological or general disease?
- (3) If neurological, is it a recurrent disturbance of function of "idiopathic" type, or is it a manifestation of a progressive process?

A safe guiding principle is that all convulsive phenomena in adults are caused by progressive pathology *until proved otherwise*. Questions should be directed both to the patient (if able to respond) and to an eye-witness, and should aim at obtaining as complete a picture as possible of the attack with its prodromal and post-ictal phases. Answers should be sought to the following questions:—

- (a) Is this the first attack?—if not, when did they begin and how often do they occur?
- (b) Are the convulsions generalised from the outset, or do they begin in one particular area and spread?
- (c) Are they accompanied by unconsciousness, preceded by any special aura, or followed by any particular abnormalities, mental or physical?

- (d) Has the patient been complaining of symptoms of general disease recently, or did the convulsion occur unexpectedly?
- (e) Have there been any symptoms to suggest that the convulsive attack is simply an episode in a progressive neurological disorder such as tumour, arterial disease, or luetic affections? If a female, is she pregnant?

<i>Hysterical fits.</i>	<i>Epileptic fits.</i>
Follow an emotional crisis.	Fairly constant periodicity day and night.
No incontinence.	Incontinence.
Patient not hurt.	Tongue biting and sometimes injury from falling.
Movements spectacular, no clonic or tonic sequences.	Tonic and clonic phases.
Corneal reflexes present.	Definite march of events.
	Corneal reflexes absent.
	Conjugate deviation of the head and eyes.
Plantar responses absent or flexor.	Plantar reflexes may be extensor.
Attack may be prolonged to impress spectators.	Attack of short duration.

FIG. 22—Hysterical and epileptic fits compared.

- (f) Is there a family history of fits?
- (g) Has the patient suffered any recent injury which might cause either cortical scarring or tetanus?
- (h) Is there any evidence of intoxication of alcoholic or other type?

Most patients are unconscious for at least a few minutes after a fit, although the focal type of attack may not be accompanied by any loss of consciousness. Any difference in reflexes, motor power, and sensation on the two sides of the body should be noted. Both plantar responses are commonly extensor for a brief period after the convulsion, but any difference in the two sides may suggest the presence of a focal lesion. Focal attacks are frequently followed by weakness of the parts affected (Todd's palsy). With organic lesions this weakness may become progressively

worse in each attack until complete paralysis may develop. This is not the case in idiopathic epilepsy.

As in the case of unconsciousness, *always take the patient's blood pressure and test the urine.* Examine the heart with particular attention to disturbance of rhythm.

When the attack is over, consideration should be given to its possible cause, since this may call for further urgent treatment. A list of causes of epileptiform attacks in adults is therefore given.

Causes of epileptiform attacks.

IDIOPATHIC.

1. Major epilepsy.
2. Minor epilepsy.
3. Other special varieties of epilepsy (*e.g.*, myoclinic).

SYMPTOMATIC.

1. Cerebral tumour (including abscess).
2. Cerebral syphilis.
3. Cerebral arterial disease
 - (a) Arteriosclerosis.
 - (b) Thrombosis.
 - (c) Embolism.
 - (d) Vasospasm.
 - (e) Hypertensive attacks.
4. Traumatic epilepsy.
5. Encephalitis.
6. Malaria.
7. "Toxic" convulsions. (Uræmia, acute nephritis eclampsia, lead poisoning, acute hepatic necrosis).
8. Stokes-Adams attacks.
9. Asphyxia.
10. Hypoglycæmia.
11. Intracranial cysticercosis.
12. Strychnine poisoning.
13. Tetanus.
14. Hysteria.

Treatment of status epilepticus.—This demands special treatment since, if not arrested, the continuous fits may cause death from exhaustion. It is liable to be precipitated by the *sudden* stoppage of sedative drugs in an epileptic patient. The following plan of treatment is recommended. Since paraldehyde is always

available and is usually rapidly effective on intramuscular injection, this should be used first (*see page 597*). The adult dose is 6 to 12 ml. repeated in four to six hours if necessary. (For a child 2 to 6 ml. may be used according to age). Alternatively hexobarbitone 0.5 to 1.0 G. in 10 per cent. solution intramuscularly may be used and repeated if necessary every four to six hours. If these prove unsuccessful intravenous thiopentone or even chloroform anaesthesia should be used. After the status has settled intramuscular sodium phenobarbitone gr. 3 every four hours should be given for the next 24 hours, and subsequently oral phenobarbitone in the doses required.

A common emergency problem is to decide whether a "fit" is organic or hysterical in nature. The table (*Fig. 22*) may be helpful in making this distinction but it is always wise to assume that there is an underlying organic disturbance.

SUDDEN PARALYSIS

This may affect one limb (monoplegia), the face, arm and leg on one side (hemiplegia), both legs (paraplegia), or all four limbs (quadriplegia).

HEMIPLEGIA

Sudden hemiplegia may result from:—

1. Cerebral thrombosis (including syphilitic arterial disease).
2. Cerebral hæmorrhage (including bleeding from the middle meningeal artery (extra-dural), and hæmorrhage into a cerebral tumour (*page 198*)).
3. Cerebral embolism (*page 208*).
4. Encephalitis (*page 198*).
5. Trauma (*page 194*).
6. Hypertensive encephalopathy and vaso-spasm.
7. Hysteria.

Cerebral thrombosis.

This has many causes and may occur at any age but is most often seen in elderly subjects with arterial disease. The clinical picture varies with the site and its underlying cause. The onset is relatively slow and may be accompanied by unconsciousness or convulsions. The full picture may take 24 to 48 hours to develop.

Treatment.—Lumbar puncture should be performed. A clear fluid under normal pressure supports the diagnosis of thrombosis

and is against that of hæmorrhage. If the patient is unconscious the appropriate routine of nursing and feeding should be carried out. On no account should venesection be performed, or hypertonic fluids administered. If the diagnosis is certain anti-coagulants may be given (*page 595*). Papaverine 65 mg. (gr. 1) subcutaneously every four hours should be given both for its sedative and vasodilator effects. Pain and restlessness call for sedatives and analgesics but care should be taken to avoid over-dosage with barbiturates and other depressants.

Cerebral embolism.

Since embolism can be diagnosed more confidently if the source is known, a list of possible causes is given:—

1. Thrombosis in any of the large arteries from which the cerebral arteries arise, including the aorta (aneurysm).
2. Mitral and aortic endocarditis.
3. Mural thrombosis following cardiac infarction.
4. Mitral stenosis with left auricular thrombosis.
5. Thrombosis of a pulmonary vein.
6. Pulmonary infection or neoplasm.
7. Fractures (fat embolism) (*page 135*).
8. In 4 per cent. of cases of cerebral embolism, the embolus originates in a systemic vein and reaches the left side of the heart through a congenital opening in the auricular or ventricular septum ("crossed" or "paradoxical" embolism).

Symptoms are of rapid onset and vary with the site of lodgment. Consciousness is not usually lost completely but the patient is dazed and apprehensive. There may be further emboli from the original source.

Treatment.—Symptoms often diminish quickly. If they increase, thrombosis is probable and administration of anti-coagulants (*page 595*) must be considered. Papaverine 65 mg. (gr. 1) should be given subcutaneously every four hours. The underlying condition should be treated when practicable, *e.g.*, ligation of a systemic vein where thrombophlebitis is suspected to be the cause of embolism. The value of stellate ganglion block remains undecided but if the diagnosis of embolism or thrombosis rather than hæmorrhage is established it is worthy of trial as it is a simple procedure without harmful complications. (For technique *see page 528*).

CEREBRAL VASO-SPASM.—The doubtful concept of a spasm of cerebral arteries was introduced to explain cases of hemiplegia with complete recovery within 24 hours. The emergency features are the same as those of embolism and thrombosis, whilst the transient nature of the symptoms may make specific treatment unnecessary. As in the case of embolism and thrombosis papaverine is recommended. The patient usually benefits from reassurance and mild sedatives.

PARAPLEGIA AND QUADRIPLEGIA

Paralysis of the limbs may be spastic (with increased resistance to passive movement, increased tendon jerks, extensor plantar responses, and knee and ankle clonus), or flaccid (with diminished or absent tendon jerks and flexor, or absent plantar responses).

Spastic paraplegia is caused by a lesion of the upper motor neurone, whilst flaccid paralysis results from lower motor neurone lesions which may be in the anterior horn cells, the peripheral nerves, or the muscles. An exception to this generalisation is that the paralysis resulting from acute upper neurone affections is flaccid at first and it is only later that the characteristic spastic condition develops. The causes of sudden paralysis may be:—

A. IF SPASTIC (*e.g.*, spastic paraplegia, or flaccid paraplegia becoming spastic).

1. Trauma (fracture dislocation of the spine).
2. Hæmatomyelia.
3. Transverse myelitis.
4. Caisson disease.
5. Hysteria.

B. IF FLACCID

1. Poliomyelitis.
2. Polyneuritis.
3. Myasthenia gravis.
4. Familial periodic paralysis.

Fracture dislocation of the spine.

The diagnosis of this condition depends on the history of trauma in which the mechanism may be one of the following:—

- (a) Penetrating wounds.
- (b) Sudden extension of the head on the neck as in judicial hanging.

- (c) Blows on the head and shoulders. *Note.*—Spinal injury may be sustained with head injury from diving into a swimming bath or even from rising to the erect position and striking some projecting object. Such cases are often overlooked since the principal injury is at the site of contact.
 - (d) Falling from a height on to feet or buttocks. (A frequent cause of vertebral injuries amongst parachutists).
 - (e) Birth injury in infants.
 - (f) Spontaneous fracture dislocation in tuberculous or neoplastic affections of the vertebrae.
4. The fracture may result in concussion, contusion, or laceration of the cord. It is interesting to note, however, that injury to the cord, especially in the cervical region, is not an inevitable sequel of vertebral damage.

Symptoms and signs.—Complete interruption of the cord leads to flaccid paralysis later becoming spastic, and loss of all sensation with paralysis of the bladder and rectum. If the lesion is in the upper cervical region it is usually fatal because of paralysis of the intercostal muscles and diaphragm. As spasticity replaces flaccidity the patient regains a measure of control over his organic reflexes and emptying of the bladder and rectum is no longer automatic.

Concussion and contusion of the spinal cord may result in equally severe *immediate* disturbance, but show early evidence of recovery of function. When damage occurs below the first lumbar vertebra the cauda equina alone is affected, producing disturbance of bladder and rectum, variable weakness of the legs, and loss or diminution of tendon reflexes with associated sensory loss, according to the roots principally affected.

Prognosis clearly depends on the site and extent of the damage. There is no hope of recovery of function following complete section, but the patient's general health can be maintained. In incomplete lesions the degree of recovery can usually be forecast as soon as spinal shock passes off (in two to four weeks).

Treatment.—The consensus of opinion is that conservative treatment is best. Only in cases where there is evidence of gross deformity or a foreign body causing compression can any hope be held out of improvement from surgical intervention. At other times, the development of severe root pains caused by compres-

sion may require surgical relief. (*For an account of the general treatment of a paraplegic case, see page 216*).

Hæmatomyelia.

Bleeding into the spinal cord may occur in acute inflammatory lesions, toxic states, blood diseases, and injuries. Congenital angiomata and syringomyelia may predispose. The commonest site of the hæmorrhage is in the grey matter of the cervical bulb, extending upwards and downwards for a few segments.

There is usually a fairly rapid onset of progressive muscular weakness in the upper limbs and signs of lower motor neurone disturbance, with a lesser degree of spastic paralysis in the lower limbs. The clinical picture may be obscured by spinal shock at the onset and occasionally the presenting symptom is respiratory distress. Paræsthesiæ are not uncommon. Involvement of the spino-thalamic tract often results in dissociated anæsthesia in addition to paralysis.

Mortality from hæmatomyelia is low. The causes of death are respiratory paralysis, urinary infection or other complications of the paraplegic state.

Treatment.—The patient must rest in bed. Mental tranquillity and sleep must be secured with suitable hypnotic drugs. A respirator should be at hand (*page 541*). Appropriate general care should then be instituted (*page 216*).

Myelitis.

The spinal cord may be affected by a number of acute pathological processes, and the term myelitis is applied to the sudden development of cord deficit. The causative agents include those which also produce encephalitis (including suppuration), disseminated sclerosis, Devic's disease (retrobulbar neuritis and transverse myelitis) tuberculosis and occasionally syphilis. In certain other cases the acute "myelitis" may result from the sudden expansion of an angiomatic malformation of the spinal cord.

Myelitis occurs typically in a young adult. Marked paraplegic symptoms develop in a matter of hours, usually with pyrexia. Pain in the back is accompanied by a girdle sensation at the affected level and often subjective sensory disturbances below. The paraplegia is at first flaccid; tendon reflexes are absent and there is retention of urine and fæces. Later, spasticity

usually supervenes. The disease may ascend and cause dangerous respiratory embarrassment. Cerebrospinal fluid changes may at first be negligible though pleocytosis and elevation of protein are common. Froin's syndrome (yellow fluid with a high protein content) may appear later.

Treatment.—This depends on accurate diagnosis and the application of appropriate specific measures, *e.g.*, for syphilis and the pyogenic infections and the correct management of the paralytic state (*page 216*).

The prognosis, which is of the utmost importance to patient and relatives, is somewhat variable. The *immediate* risks include respiratory weakness, urinary sepsis, and trophic sores. To a considerable extent, expectation of recovery depends on the specific cause. As a rough guide it may be said that the earlier recovery begins, the better the final result. Even following prolonged paralysis, function may return in some degree with at least ability to stand, and to walk about in the house.

Caisson disease. (*page 375*)

The paralytic manifestations of hysteria.

Many doctors are reluctant to make a diagnosis of hysteria unless there has been a long history of recurrent minor complaints of a non-specific nature, especially in women of menopausal age. Once the diagnosis is made, however, there is a danger of every subsequent symptom being attributed to it.

When hysteria is diagnosed there must be no evidence of organic disease. An extensor plantar response particularly invalidates any suggestion of a purely functional disturbance. Similarly, well-marked, sustained nystagmus, pupillary and optic nerve abnormality, or muscular fibrillation indicates the presence of organic disease. Changes in tendon reflexes (though not total loss), wasting and contractures, may, however, result from hysteria.

Hysteria should not be diagnosed simply by exclusion. A positive psychiatric disability should be established and it is not sufficient for the patient to be of chronic psychoneurotic disposition. Injury (particularly head injury) may act as the trigger producing hysteria and often serves to localise the site of paralysis.

The hysteric is highly suggestible and presents paralytic manifestations of a wildly improbable nature. Thus there may be

paralysis of the movements of a single joint which cannot be caused by organic nervous disease. With appropriate suggestion it is usually easy to demonstrate sensory changes totally at variance with anatomical and physiological considerations. The paralysis, too, has often certain anomalous features. Whether spastic or flaccid, passive movement is usually resisted with considerable force—unlike any organic paralysis. If the legs are affected, the gait betrays abnormalities of an unusual nature. When walking, the hysterical subject does not circumduct the “spastic” leg but drags it laboriously along the ground. The hysterical paraplegic patient makes Herculean efforts to stand and move, but the absurd incoordination and extravagance of his movements betray his desire to make himself conspicuous by his failure rather than by his success.

Acute anterior poliomyelitis. (*see also pages 302 and 566*).

This acute febrile condition is caused by a virus and results in loss of power and subsequent rapid wasting of one or more groups of muscles. Its maximum incidence is in the age group two to five years, but no age is exempt.

Subclinical and abortive types are described but obviously these will not be emergencies. We are concerned here with the cases in which actual paralysis develops. Two stages are usually recognisable, and if they occur they aid the diagnosis. Either may occur without the other.

1. The preparalytic stage is characterised by pyrexia, gastrointestinal disturbance and headache. The patient is miserable, restless, and hypersensitive to touch. Meningeal signs may be present.
2. The paralytic stage begins after three to five days with sudden paralysis in one or more groups of muscles. At first all four limbs may be paralysed but subsequent permanent paralysis tends to affect one limb. Occasionally transitory sensory signs are present. Involvement of respiratory movement is a grave complication.

The C.S.F. shows a raised cell count to between 10 and 1,000 (usually 50 to 250) per cu.mm. The cells are at first polymorphs but later lymphocytes. Protein rises to 100 mg. per 100 ml. and there may be a mild second phase elevation of the Lange gold curve.

MANAGEMENT.—The emergency management of a case of poliomyelitis has two aspects, namely, the public health problems raised and the care of the patient himself.

PUBLIC HEALTH MEASURES.—An outbreak of poliomyelitis is usually the signal for the development of acute anxiety in all concerned. Fear of its immediate and long-term effects, coupled with the publicity given to the disease, have naturally induced emotional attitudes which do not aid in management. The situation has been further complicated by the controversial evidence as to the mode of infection and portal of entry. In the light of present knowledge it is safe to assume that the disease may enter via the nasopharyngeal and gastro-intestinal routes and therefore the recommendations we make are as follows:—

1. Institute complete barrier nursing for 28 days.
2. Spray the room with D.D.T. (Dicophane, B.P.) to prevent insect contamination.
3. Dispose of faeces without danger of contamination (*page 323*).
4. Isolate child contacts for 21 days. (The estimated incubation period is 12 days). It must be remembered that persons capable of transmitting the disease may never show signs of infection themselves. If the outbreak occurs at a residential school or other institution, it is not advisable to send the children home since this opens the possibility of spread to younger and more susceptible children. In hospital no isolation of adult contacts is necessary, but it is wise to prevent contacts from taking violent physical exercise during the observation period.
5. In epidemics consider the use of convalescent serum 10 to 20 ml., immune horse serum 10 ml. or gamma globulin 1 G. (500 mg. if under 1 year) for the protection of child contacts. Nurses and students in contact with early cases may be protected by 1.5 G. of gamma globulin. Experimental work on active immunisation and nasal spraying has provided no evidence in favour of these methods for urgent prophylaxis but the sera recently evolved and now undergoing trials in the United States seem to offer good passive immunisation.

CARE OF THE PATIENT.—In the acute phase, *i.e.*, before paralysis, general nursing attention will be required. Analgesics should be given for pain. Gentle passive movements of the limbs are helpful and hypertonic saline baths are soothing for hyper-

æsthetic limbs. Foot boards should be applied to prevent early footdrop. The cabinet respirator should be made ready (*page 564*). For emergency measures in bulbar poliomyelitis *see page 566*.

If paralysis has occurred, the part affected should be immobilised in a position which will avoid over-stretching of the paralysed muscles and especially the deltoids, the small muscles of the hands, the rotators of the hip, the extensors of the hip and knee and the dorsiflexors of the foot. Retention may develop and persist for some days. It should be treated as outlined on *page 216*.



FIG. 23
The myasthenic face.

Acute infective polyneuritis.

In this condition, a short period of pyrexia is followed by a spreading flaccid muscular paralysis, pains and paræsthesiæ. Examination reveals tender muscles, diminished reflexes and hyperæsthesia or anæsthesia of glove and stocking type. The cerebro-spinal fluid shows a raised protein content (100 to 400 mg. per 100 ml.) with little change in cell content ("dissociation albumino-cytologique").

Involvement of the vagus may cause troublesome tachycardia. Respiratory paralysis may also be an urgent symptom and call for the use of the respirator. Recovery is the rule, but occasionally a patient is seen whose condition deteriorates steadily until death occurs from respiratory failure. Recent work suggests that A.C.T.H. (corticotrophin) can produce striking and rapid improvement in severe polyneuritis of this type. The dose is 25 mg. in 500 ml. of 5 per cent. dextrose, given by intravenous drip over a period of 8 hours every day for 7 or 10 days.

We have also obtained an excellent response with intramuscular injections of 40 units (mg.) of long acting A.C.T.H. gel. (Acthar gel. Armour) daily for 7 to 10 days.

Myasthenia gravis.

A case of myasthenia gravis (often misdiagnosed as psychoneurosis) may be seen for the first time in a crisis with cardiac and respiratory failure imminent.

The myasthenic face (Fig. 23) (drooping jaw, bilateral ptosis and wrinkled forehead), together with a history of being easily fatigued should suggest the diagnosis. Prostigmin (neostigmine) 2.5 mg. with atropine 0.65 mg. (gr. $\frac{1}{100}$) should be given subcutaneously at once and repeated as necessary.

Artificial respiration, and emergency treatment for pneumonia may be needed, together with nasal and parenteral feeding. After the immediate crisis is over, Prostigmin (neostigmine) can be given orally—15 mg., three, four or more times daily.

Familial periodic paralysis.

Like myasthenia gravis, this rare disease is likely to be mistaken for hysteria or a psychoneurosis. Its pathogenesis is obscure but it is thought to originate in a disorder of sodium and potassium metabolism. The victim suffers periodically from bouts of flaccid paralysis, lasting from three hours to four or five days. Its onset is characteristically in the teens, but occasionally it occurs in early childhood.

Paralysis usually appears soon after waking and may have been preceded by stiffness, hunger and thirst or other prodromata on the previous day. Precipitating causes are cold, starvation, alcohol, fatigue and menstruation. The paralysis reaches its peak in one or two hours, and is usually symmetrical but may be of hemiplegic or paraplegic distribution, and occasionally the respiratory muscles are involved. Unlike myasthenia gravis, the movements of the eyes, face and swallowing muscles are not affected.

The history of previous attacks and the familial evidence should point to the diagnosis. Examination reveals a flaccid paralysis with diminished reflexes, in contrast to hysteria where they tend to be brisk. Two other features help to differentiate the condition from hysteria: the blood potassium is low (normal 20 mg. per 100 ml. (range 14 to 22) or 5 mEq/litre (range 3.6 to 5.6)) and the muscular responses to galvanism and faradism are diminished or lost."

Treatment.—Potassium (*see page 466*) should be given by mouth and repeated if necessary. A respirator may be needed.

EMERGENCY TREATMENT OF PARAPLEGIC CONDITIONS

Having made a diagnosis of paraplegia or hemiplegia for which there is no specific treatment, it is not sufficient to rest content with "general nursing care." The prognosis depends on proper management as soon as the emergency state begins.

Some of the factors determining the outcome, such as the extent of the initial motor weakness and sphincter disturbances, are not influenced by treatment. Urinary and pulmonary infections, pressure sores, contractures and the extent of residual loss of function can undoubtedly be influenced by correct treatment.

Sphincter disturbances.

Retention of urine is the commonest sphincter disturbance calling for urgent treatment.

1. If retention results from tabes dorsalis or is of recent origin and unaccompanied by widespread neurological signs, Carbachol B.P. 1 ml. intramuscularly should be used. (Beware of over-dosage, and never use it except in solutions from an ampoule. *It must never be given intravenously*).
2. If retention is part of the picture of coma, repeated catheterisation is the best course.
3. Retention complicating serious disease of the cord, such as compression or degenerative and inflammatory processes (including poliomyelitis), demands prompt treatment. Slow decompression by catheter is the first step, followed by the establishment of tidal drainage (*page 535*). Suprapubic drainage has no place as an immediate measure.

Urinary infection.

Retention and instrumentation tend to cause bladder infection with hypogastric pain, malaise and swinging temperature and an obviously infected urine. The causative organisms should be identified, and appropriate treatment by a sulphonamide (*see page 599*), or antibiotic (*see page 601 et seq.*) instituted. We find chloramphenicol 0.5 G. twice a day to be the most efficient drug in combating infection of the paralysed bladder.

Prevention of bed sores.

Although commonly regarded as one of the long-term effects of chronic disease, the acute neurological bed sore resulting from a combination of trophic changes, local pressure, and pressure occlusion of vessels, can develop and spread at remarkable speed. Its prevention is a matter of great importance. The patient should be nursed alternately face up and face down, on rows of pillows three deep, allowing the bony prominences to lie between them. The position may be changed hourly or more frequently if the patient so desires, and if sufficient nurses are available.

Redness of the skin which disappears on pressure is the earliest sign of an impending bed sore. The usual measures of sponging, drying, and powdering, should be assiduously employed and followed by the application of astringent preparations which harden the skin, *e.g.*, equal parts of the Solution of Lead Acetate

B.P. and Tincture of Catechu B.P. If sores develop in spite of all precautions, the use of penicillin-sulphathiazole powder (500 units of penicillin to one gramme of sulphathiazole) often promotes healing.

Contractures.

The battle for recovery of function and avoidance of contractures begins as soon as paralysis occurs. Affected parts should be splinted in the position of physiological rest with particular attention to foot and wrist drop. As soon as the patient is able to co-operate, passive movements, and as many active movements as possible, should be performed. Specific actions should be practised *hourly* (and not twice weekly). Electrical stimulation of centrally paralysed muscles may be carried out with a view to maintaining function until eventual recovery of the motor neurone.

The patient should be encouraged to take gentle exercises at the earliest opportunity. Active use of the limbs is the best form of physiotherapy, and it is at this stage that the patient acquires trick movements which often minimise his disability to a remarkable degree.

PAIN AS A NEUROLOGICAL EMERGENCY

Four questions should be asked about any pain:—

Where is it? (Site and radiation).

What is it like? (Character and intensity).

What makes it worse?

What makes it better?

While we are here discussing only neurological diseases, it must be borne in mind that in practice this arbitrary grouping does not exist, and that severe pain may be other than of neurological origin. Even patients with known nervous disease may have appendicitis, and gastric ulcers are commoner than gastric crises.

Headache.

The degree of anxiety and consequent apparent urgency caused by any painful condition varies with the patient's psychological make-up. We must not be misled by the impassiveness of the phlegmatic patient on the one hand, or the anxiety of the nervous patient on the other, remembering that the significance of

a pain varies inversely as the richness of its description. This is particularly important in evaluating the significance of headache.

Severe headache presenting as an emergency may be a symptom of the following conditions:—

(1) ANXIETY AND HYSTERIA.—Headache in these conditions is often described as “terrible” or “agonising” but is usually qualified by the terms “pressure” or “bursting.” The immediate point in management is to exclude organic causes and then to approach the problem confidently as a psychoneurosis. The ophthalmoscope should always be used.

(2) SUBARACHNOID HÆMORRHAGE.—(*page 190*).

(3) MENINGITIS.—(*pages 200 and 312*).

(4) INCREASED INTRA-CRANIAL PRESSURE FROM ANY CAUSE.

(5) MIGRAINE.—The headache of migraine, especially when it occurs for the first time, may terrify the patient and call for judgment in diagnosis and treatment. Characteristically the attack begins with a visual aura, black spots before the eyes, hemianopia (to the patient's great distress at first) and fortification spectra. There may be a sensory aura of tingling in the limbs and face. Not infrequently, there is weakness also. After ten to twenty minutes the crushing headache develops, usually unilateral, but occasionally of general distribution. Nausea and vomiting may follow.

The duration of the attack may be from one hour to an entire day. On examination, the hemianopia may be detected and an associated spasm of the retinal arteries seen. The nature of the condition is probably a vascular spasm followed by dilatation, but it should be remembered that similar symptoms may result from aneurysm, hypertension and uraemia. Ophthalmoplegia may occur in true migraine but suggests very strongly the possibility of aneurysm or tumour.

Treatment.—Once the attack is well under way, little can be done, but certain drugs may be of value in the early stages. The following are alternatives:—

- (a) Ergotamine tartrate (Femergin) 1 mg. sublingually, followed by 1 mg. in one hour. It should not be given to pregnant women.
- (b) Femergin, 1 mg. subcutaneously.

- (c) Benadryl (diphenhydramine), 100 mg. in capsules by mouth followed by 50 mg. in two hours. The patient should be advised to rest.
- (d) Injection of Adrenaline B.P. 0·3 to 0·6 ml. (5 to 10 m) subcutaneously.

Pain in the trunk (root pains).

Not infrequently one sees in the neurological out-patient clinic, a patient in such acute agony that he beseeches the doctor to give him relief, and threatens suicide if nothing is done to abolish his pain. Such unfortunate people present problems both of immediate therapy and long term diagnosis. Careful questioning elicits the fact that the distribution of the pain corresponds anatomically to root distribution and is usually accentuated by coughing and sneezing. The possible causes are tabes dorsalis, secondary carcinomatous deposits, extramedullary spinal tumours, Pott's disease, spondylitis, meningeal inflammation, herpes zoster—in the pre-eruptive stage, and hysteria.

The characteristics of the pain should indicate the diagnosis, but the following points may be useful:—

- (1) Girdle sensations of tabes dorsalis will be accompanied by corroborative signs in the pupils and tendon reflexes.
- (2) Severe root pains in an elderly person, in the absence of signs of syphilis, are usually caused by secondary neoplastic deposits.
- (3) The presence of kyphosis suggests tuberculous disease of the spine.
- (4) If the location of pain and altered sensibility do not coincide with root distribution, the condition is either attributable to general disease or to hysteria. Visceral referred pain often assumes a root distribution and must be considered.
- (5) It must not be forgotten that patients with neurological disease sometimes have appendicitis or perforated ulcer.

Treatment.—Having excluded a surgical cause it is usually necessary to give symptomatic treatment. Whilst realising the dangers of addiction we feel that too often a patient is allowed to suffer untold agonies for the want of adequate analgesia. In painful incurable disease with a short prognosis—such as cancer with metastases—the correct dose of analgesic is *that which relieves*

R.	Heroin	11 mg. (gr. $\frac{1}{8}$)	
	Aspirin	0.32 G. (gr. 5)	Make a powder.
	Phenacetin	0.32 G. (gr. 5)	
	One powder every four hours as required.				

Visceral pain of neurological origin.

Treatment.—For relieving laryngeal crises, amyl nitrite is usually satisfactory. Gastric crises are best treated by injections of morphine 16 mg. (gr. $\frac{1}{4}$) or physeptone 2.5 to 10 mg. Intramuscular sodium phenobarbitone 0.2 G. (gr. 3) repeated after four hours if necessary will help to relieve pain and promote sleep.

Peripheral nerve disease, including sciatica, spinal cord degeneration and neoplasms, and affections of the thalamus may at times cause very severe pain for which adequate analgesics should be prescribed pending diagnosis. As alternatives to morphine and heroin the following prescriptions are helpful:—

- | | | | |
|-----------------------|-----|-----------------------------|----------------------------------|
| 1. Phenacetin | ... | 0.32 G. (gr. 5) | As a powder every
four hours. |
| Aspirin | ... | 0.32 G. (gr. 5) | |
| Codeine | ... | 32 mg. (gr. $\frac{1}{2}$) | |
| 2. Sodium phenobarb. | | 45 mg. (gr. $\frac{2}{3}$) | One ounce four times
a day. |
| Sodium bromide | | 0.65 G. (gr. 10) | |
| Phenazone | ... | 0.65 G. (gr. 10) | |
| Tincture of Gelsemium | | 1.2 ml. (m. 20) | |
| Chloroform water | | 30 ml. (1 fl.oz.) | |
| 3. Codeine | ... | mg. 30 | Every four hours. |
| Pethidine | ... | mg. 50 | |

4. Disephonine (Physeptone) 2.5 to 10 mg. by mouth or by injection.
5. Levorphan (Dromoran) 1.5 mg.,
Chlorpromazine (Largactil) 25 mg. Every four hours.

✓ VERTIGO

Sudden severe vertigo may call for urgent treatment. Although the sense of position can be upset by many factors, severe vertigo results only from an irritative lesion in the labyrinth and its associated structures, the cerebellum and the association pathways.

If more than merely first-aid measures are contemplated the problem should be tackled as follows:—

- (1) Take a detailed history.

Is this the first attack or one of a series? Recurrent attacks in a middle-aged person suggest Ménière's syndrome. Migraine and epilepsy may have a vertiginous aura. Remember too that acoustic nerve tumours may cause recurrent vertigo for some time without other signs. Head injury may result in vertigo after months or years. A first attack suggests the additional possibilities of disseminated sclerosis, syringomyelia, posterior inferior cerebellar artery thrombosis and ear disease.

Is there a history of previous ear infection or evidence of it now?

Is there a history of other periodic nervous upset?

Is the patient taking drugs (quinine, salicylates, alcohol, streptomycin)?

Is there tinnitus? This suggests a peripheral origin.

- (2) Examine the ears for wax and suppuration. Rarely, small vesicles in the external auditory canal or throat and a seventh nerve palsy will be found, indicating herpes of the geniculate ganglion.

- (3) Carry out hearing tests. If the patient is deaf to speech and watch tests, use a tuning fork (C.256) to distinguish nerve (perceptive) from middle ear (conductive) deafness.

Weber's test. Place the tuning fork on the patient's forehead and ask him where he hears the sound. In middle ear deafness the sound is heard in the affected ear. In nerve deafness it is heard in the normal ear.

Rinné's test. Place tuning fork on the mastoid process and occlude the ear. As soon as the patient ceases to hear it, quickly transfer the prongs to the meatus. In middle ear deafness he will hear no sound (i.e. bone conduction is better than air conduction). A normal or partially nerve deaf person will still hear the sound (air conduction is better than bone conduction)

Vertigo with normal hearing.	Vertigo with middle ear deafness.	Vertigo with nerve deafness.
Disseminated sclerosis.	Otitis media.	Ménière's syndrome.
Migraine	Otosclerosis.	Acoustic nerve tumours
Epilepsy	Trauma.	Labyrinthitis.
Vascular Diseases.		Trauma.
Syringomyelia.		Geniculate herpes.
Trauma.		
Tumours	Intra-cranial extension of ear disease may cause vertigo with either middle ear or nerve deafness. Drugs causing vertigo may or may not also cause deafness.	
Hysteria.		

FIG. 24

Causes of vertigo in relation to hearing.

The causes of vertigo in relation to hearing are shown in Fig. 24.

(4) Examine the nervous system.

Attention should be directed to the possibilities of disseminated sclerosis, syringomyelia and tumours and vascular lesions of the brain stem and cerebello-pontine angle. Any condition resulting in increased intracranial pressure may cause vertigo but this will rarely be a primary symptom. From the emergency point of view the only likely cause under this heading is **posterior inferior cerebellar artery thrombosis**. In this condition a wedge-shaped infarct on the side of the medulla causes a characteristic syndrome with severe vertigo and inco-ordination. On examination there is Horner's syndrome (miosis, ptosis and enophthalmos) and paralysis of the palate, pharyngeal muscles and vocal cord on the side of

the lesion. The face on this side shows loss of pain and temperature sense and these are lost also in the limbs and trunk on the opposite side.

- (5) If the diagnosis is still in doubt, incipient meningitis, arteriosclerosis of the labyrinthine vessels and hysteria should be considered.

Treatment.—The immediate problem is the relief of the giddiness and the associated vomiting. The patient will usually have taken to bed. Specific therapy depends on the diagnosis.

- (i) Ménière's syndrome. Try chloretone (Chlorbutol B.P.) 0.65 to 1.3 G. (gr. 10 to 20) and repeat in six hours supplemented by Benadryl (diphenhydramine) 100 mg. if necessary. In very severe attacks phenobarbitone sodium 0.2 G. (gr. 3) should be given. Occasionally morphine 16 mg. (gr. $\frac{1}{4}$) with hyoscine 0.32 mg. (gr. $\frac{1}{200}$) by injection is needed.
- (ii) Migrainous vertigo should be treated by ergotamine tartrate 1 mg. intramuscularly.
- (iii) Infective conditions call for penicillin (*page* 601) or other antibiotics (*page* 605 *et seq.*) and sulphonamides (*page* 599).
- (iv) For vascular causes papaverine 65 mg. (gr. 1) subcutaneously should be used as it combines the advantages of sedation and vasodilatation. Anti-coagulant therapy (*page* 595) is indicated in cerebellar artery thrombosis.
- (v) For intracranial tumours phenobarbitone is best. Morphine should not be used because of its depressant effect on the respiratory centre.

FERGUS R. FERGUSON.

LAURENCE A. LIVERSEDGE.

CHAPTER XII

*Psychiatric Emergencies**

PSYCHIATRIC emergencies occur when a patient is creating a social disturbance owing to illness or when psychiatric disorder is seriously impairing his bodily state and ordinary measures for checking it are refused or impractical. The primary cause may be a somatic illness, a frank psychosis or a severe neurotic reaction. Accurate psychiatric diagnosis is not needed at once and here we will consider only the methods of dealing with emergency situations.

These may be divided into two types (1) those calling for immediate drastic action and removal of the patient from home. This group includes acute mania, delirium and psychotic excitement; acute suicidal gestures; post-epileptic furore; severe confusional conditions or any state in which the patient is in danger of injuring himself or others. (2) Those where the social disturbance is less but the patient's position precarious because of his behaviour or his physical well-being.

Examination.

At the interview it is most desirable that one relative is seen *alone* and that as coherent a picture as possible is obtained before the patient is examined. At the examination no more than one person besides the doctor should be present. It should be as full as possible for, although the psychiatric disturbance is clear, there may be a somatic cause.

Urgent restraint and sedation.

While few should be present at the examination *as many as necessary* should be called in if force is needed. We do the patient harm by summoning too few so that he runs hither and thither harming himself and them. An acutely maniacal man may require six or more persons to sit on him before he can be given an injection. When the patient sees the assistants he will realise that the doctor's course of action is going to be followed willy nilly.

* (For Psychiatric Emergencies (i) on board ship *see page 380*, (ii) in pregnancy *see page 109*, (iii) in the gynæcological patient *see page 113*.)

If he has to be held down, see that he lies on his face so that any injection is given into his buttock. If the patient is co-operative enough to take medicine by mouth give amylobarbitone 0.5 G. (gr. $7\frac{1}{2}$) or paraldehyde (*page 597*). Failing this inject (with a long needle through the clothing if need be) paraldehyde (*page 597*) or morphine 32 mg. (gr. $\frac{1}{2}$) and hyoscine 0.2 mg. (gr. $\frac{1}{75}$). Large doses of morphine, say 65 mg. (gr. 1) can be safely given and do not cause respiratory depression if combined with amiphenazole (Daptazole) 15 mg.

Removal.

The "duly authorised officer" (D.A.O.) (Mental Welfare Officer—formerly Relieving Officer) is the person empowered to remove a patient against his will. The police will give his telephone number (in London it is Waterloo 5000). He requires a short written statement of the doctor's findings and opinion. If he agrees, and he is almost always most co-operative, he will provide conveyance and attendants. It is not necessary to wait for the D.A.O. if the written statement is left at the house.

Some relatives may protest against mental hospital treatment. If they refuse to accept advice it is wise to get them to sign a note to this effect. If the objections are social ("My husband can't go to the local bin") we can assure them that plans for removal to a "private hospital" may be made later. The essential point is the emergency. Neither the D.A.O.'s order for admission to a mental observation ward nor the urgency order constitute "certification." Neither lasts more than seventeen days and during this time the full implication can be discussed by the relatives with the psychiatrists.

There are two other ways of sending a patient urgently to a mental hospital.

(1) As a voluntary patient. This method should not be used if it is felt that the patient will not stay. The patient should sign a statement (preferably at home) to say "I, A.B., desire to enter C.D. Hospital as a voluntary patient. I undertake to follow the instructions of the Medical Superintendent and to give 72 hours' notice of my intention to leave."

(2) As a temporary patient. This method is only applicable to patients who are unable to express themselves as willing or unwilling for treatment but it is very suitable for the stuporose, confused and delirious case as well as for the elderly dement. Form

A1 (Mental Treatment Act, 1930) (*see page 3*) is completed. It consists of a request for admission from a relative and two statements by doctors who must not be partners, one of whom must be specially approved to advise about temporary patients. All specialists in mental disorders are so qualified.

LESS URGENT MENTAL DISORDERS

Other mental disorders may constitute emergencies though they are far less socially disturbing. These are

(a) States in which the patient is highly suspicious yet equally careful to disguise his beliefs. Here the danger is sudden action against others.

(b) Early deliria where suspicion and misunderstanding are marked, and which may advance rapidly because fluid balance and carbohydrate metabolism are so disturbed.

(c) States of stupor which may have a depressive, schizophrenic or organic or panic basis and where metabolic adjustments are impaired or unable to respond to the somatic crisis.

Fine judgment is needed by the doctor to sail between the Scylla of discussing the condition because he is sure the relatives are overwrought and the Charybdis of urging action because he is perplexed and uncertain. Here amylobarbitone is a great help. The patient under the influence of 0.5 G. (gr. 7½) may more readily drink glucose and the doctor and relatives may come to a better understanding.

GENERAL PRINCIPLES

Three principles can be enunciated.

(1) The first few hours in the treatment of any psychiatric emergency may be more fateful than the subsequent months. Removal to a strange environment under trained nurses may be far less disturbing than the continued attentions of well-meaning but harassed relatives. If the doctor feels he can remain in control of the family he may be right to keep the patient at home. If he believes the relatives will thrust upon him piecemeal decisions removal is better.

(2) The "damage" done by sending the patient away is exaggerated. It is surprising how seldom a patient or his relatives complain of "that other doctor" who sent him to an observation ward. Such wards are by no means the bedlams they are said to be. Gradually it is being learnt that general hospitals and mental

hospitals have much in common—above all a hopeful therapeutic outlook.

(3) Acute mental reactions have by no means a hopeless prognosis. Like all symptoms they are warnings that some action and, possibly removal is called for. Many of those suffering from confusion, delirium and psychopathic disturbances recover.

GENERAL MANAGEMENT

If the less acutely ill patient remains at home for even a few hours he should have one companion with him whether asleep or awake. The best one, failing a trained nurse, is the calmest relative or, where such exists, the old family retainer. Such a person conveys a calm which others, though well-intentioned and learned, may lack. It is strange how often it is not brains but loyalty that counts most in these cases.

Adequate fluids (four pints a day) suitably flavoured are essential. Vitamin deficiency may be present and is conveniently treated by paired ampoules of Parentrovite (Vitamins Ltd.) intravenously or intramuscularly three times a day. Sudden large doses of sedatives may be called for but repeated sedation to avoid admission to hospital confuses the picture. It is much better to be sure that the patient is at a hospital with psychiatric beds. There, drugs can be withdrawn and the clinical picture can be viewed unclouded. Bromides are always undesirable in these cases.

The successful dealing with psychiatric emergencies depends not a little on intuition and temperament. Many may take a firm line but few can keep it. It cannot be repeated often enough that quiet resolution in the doctor is all-important. Other than in those cases which need immediate removal, the doctor may long for time in which to think. Amylobarbitone gives this breathing space while the doctor sits, quietly balancing the pros and cons of the situation. If he does this or is so well-trained that he can dispense with it he will realise that his further actions should have the following aims.

(1) To still, if possible, misunderstanding in the patient's mind. Few people are so mad that they have no apprehension about what will be done to them. Some may have justifiable cause for believing that relatives are behaving incomprehensibly. An acute mental illness may have been preceded by an incubatory period in which

the patient was only dimly aware that something was wrong and some of his behaviour may be due to that. It is well to remember that patient and relatives are all facing an emergency as well as ourselves. They have no principles on which to act; emotion and reason are in a state of war. In our desire to get them to act rationally medical men are to be forgiven if they forget that the patient would be the more reasonable, could we but understand the premises of his belief. The child, whose mother had forgotten to give him the belladonna prescribed, made up for this by emptying half the bottle down his throat. Shortly after he began seeing pennies on the ground, in the air, near and far. He appeared "as mad as a hatter." But, in fact, he was describing in his own way the central scotomata that belladonna poisoning and paralysis of accommodation produced. In others, the rage, furore and behaviour disturbance may be based on misunderstanding, totally incomprehensible to the participants, and only to be understood by an outside observer—the doctor. These rages, storms, and sometimes apparently hallucinatory experiences are seen amongst children, and others with immature minds such as defectives, hysterics, psychopaths. In all such cases, the removal of the relatives may gradually calm the patient. He may respond to quiet examination by the doctor, and if this has gone well, he may then be ready to show some of the misunderstanding upon which his apparently irrational conduct is based. It may still be wise to remove him from home, but a short discussion may enable him to go willingly. A talk with the doctor may be the first hint to the psychopath that there are people in the world who can comprehend. This attitude does not imply indifference to the social disturbance caused, but it can include disapproval of that as well as hope for the future.

(2) Not only should the doctor contrive to get his plans accepted, he should visualise some of the despair in the relatives' minds. Their plans may be less irrational than they seem if we remember the blame they may attach to themselves. The facade they may have kept up is imperilled. The emergency threatens to destroy this. Their repugnance to our plans may seem irrational: if we could see the conflicts behind we might well understand better.

Dogmatic argument or bluster or anger will achieve little. It is better that we emphasise that "our experience leads us to this

advice" rather than be drawn into endless discussion. Such an attitude comes with time, but the young assistant may have it if he is grounded in human understanding.

THE RISK OF SUICIDE

The old belief that he who threatens does not carry out self-destructive gestures is far too superficial. Yet there are at least six times as many gestures as successes. The successes unfortunately include a large percentage of persons whose disorder of mind is completely curable. The gestures occur as a danger signal—a warning that the patient is asking for some steps to be taken. It is true that the patient's terms may be quite unjustified, as in some psychopaths. But it is better that they should be stated and discussed, rather than flagrantly disregarded. The writer knows of no certain way of guarding against suicide, except supervision. Many families seem incapable of comprehending what this entails. It is better to suffer ignominy for insisting upon removal rather than remorse after the second gesture became successful. Be specially cautious in cases where the patient is a lone wolf; poorly socialised; in cases with marked self-blame about the past; in cases where there is a family history of suicide (often admitted "cagedly").

MENTAL DISORDERS IN ORGANIC DISEASE

Delirium and confusion are suggestive of mental disorder occurring as a result of underlying organic disease, trauma or toxæmia. Minor head injury and epilepsy should never be forgotten.

Their prognosis is that of the underlying disease and often very good. The onset of mental symptoms may be very insidious. In delirium tremens the clouding of consciousness hardly describes the quiet, subtle changes that occur, long before "tremens" (=trembling) is evident. Early psychiatric assessment of alterations in mood, in predictability and in attention in an out-patient department may forestall emergencies. The treatment of pre-delirious states, as well as deliria requires external vigilance and adequate observation. Vitamins (*see page 228*) should be given as well as antibiotics. A delirious state can certainly continue after the temperature has fallen and there has been a satisfactory response to antibiotics. Be reluctant to blame alcohol or bromide

alone without remembering that structural damage may be occurring as well.

LEGAL ASPECTS

The doctor may wonder about legal action which wrongful certification may entail. His position has been greatly improved by the Mental Treatment Act, 1930. If he can show that he acted in good faith no action at law will be successful against him. His main anxiety is now therefore whether the patient should remain at home or not. In making his decision his best assurance against legal action is to make a full examination and to record his findings.

HENRY WILSON.

CHAPTER XIII

Medical Emergencies in Diabetes

KETOSIS

KETOSIS means the presence in the blood and urine of "ketone bodies." Of those in the urine 70 per cent. is beta-oxybutyric acid, but its presence cannot be recognised by either Rothera's (nitroprusside) or Gerhardt's (ferric chloride) tests. The remaining 30 per cent. is almost entirely diacetic acid. Acetone, though excreted in the breath, is only present as traces in fresh urine. Diacetic acid is by far the more important substance to look for and can be detected by the nitroprusside test in dilutions of 1 in 400,000. Acetone (formed when diacetic acid decomposes on standing) also gives a positive reaction but only in dilutions of 1 in 20,000. The ferric chloride test gives a positive reaction with diacetic acid but only in higher concentrations of 1 in 2,000 and is negative to acetone. Hence the nitroprusside test should be done first and followed, if positive, by the ferric chloride test. If this also is positive it indicates severe ketosis and the possibility of coma.

Symptoms.

Severe ketosis may be symptomless but is more often associated with one or more of the following symptoms: shortness of breath, anorexia, nausea, vomiting, drowsiness and abdominal pain. Vomiting is particularly important as it tends to set up a vicious circle in which starvation increases the ketosis, and this in turn aggravates the vomiting. Ketosis may then swiftly progress to coma. Abdominal pain, especially when associated with vomiting, may present a difficult diagnostic problem, the importance of which is accentuated by the fact that the unnecessary opening of the abdomen of a patient in severe ketosis may greatly prejudice recovery. The abdominal pain of ketosis is usually diffuse, rigidity is not a marked feature and signs of peritoneal irritation are typically absent (*see also page 58*). Rarely there may be marked upper abdominal distension from acute dilatation of the stomach (*see page 467*).¹

Causes.

Vomiting and starvation are dangerous in diabetics and if absence or deficiency of insulin is added coma is likely to follow. Hence diseases of the gastro-intestinal tract, particularly gastro-enteritis, stand high in the list of causes of diabetic coma, not only because they cause vomiting and starvation, but also because it is common for diabetics suffering from them quite erroneously to conclude that if they can't eat they don't need insulin. Other causes include parenteral infections such as septicæmia, pyelitis and pneumonia, and local infections such as carbuncles, infected gangrene and insulin abscess, and the stopping of insulin.

It is important to realise that, although ketosis may clear up spontaneously, it is more likely, if untreated, to progress to coma and death. It follows that every effort should be made to prevent ketosis and, if it occurs, to treat it before the ferric chloride test becomes positive. If every diabetic with severe ketosis were regarded as a potential case of diabetic coma, and appropriately treated before the onset of symptoms, the incidence of this most dangerous, but usually most preventable medical emergency, could be greatly reduced.

The treatment of severe ketosis will therefore be considered under the heading of diabetic coma.

DIABETIC COMA

There is no sharp dividing line between severe ketosis and pre-coma, and the latter term is best reserved for those cases which present clinical manifestations of ketosis but, although drowsy, retain consciousness. The term diabetic coma is often loosely applied to this latter group but should be reserved for those in whom there is actual loss of consciousness.

Diagnosis.

First obtain as detailed an account as possible of events immediately preceding the onset of coma. Often the diagnosis will then be apparent before the patient is examined. This is no less true of patients first seen in coma than in known diabetics. In the former a history of thirst, polyuria, and loss of weight preceding the onset of coma will focus attention in the right place when it comes to examination. In a known diabetic taking insulin the diagnosis is most likely to be diabetic coma or hypoglycæmic coma; the value of history in distinguishing between these two conditions cannot be over-emphasised.

The onset of diabetic coma is gradual and follows a period of severe ketosis; sudden unconsciousness without the premonitory symptoms and signs of ketosis is rarely, if ever, due to diabetic coma. The signs result from ketosis (furred tongue, acetone in the breath and deep abdominal respiration) and dehydration (lowered ocular tension, dry tongue and skin, rapid pulse and low or falling blood pressure).

The urine shows heavy glycosuria and intense ketosis, the ferric chloride test being strongly positive. Chlorides are typically diminished or absent (*see page 463*). Very occasionally there may be complete absence of sugar and ketone bodies in the urine associated with severe impairment of renal function and the passage of large numbers of casts; in such cases the secretion of urine may fall or even cease, and the blood urea reach very high figures. The blood sugar is invariably raised and usually exceeds 400 mg. per 100 ml. although diabetic coma may very rarely occur with lower levels of 300 upwards. The ketone bodies in the blood are also greatly increased and, although not usually estimated, may be detected by applying Rothera's test to a few drops of plasma or serum in a watch glass, a useful procedure in the rare cases, referred to above, in which renal damage results in absence of ketonuria.

Differential diagnosis.

If hypoglycæmia is excluded (*see page 242*), the commonest differential diagnosis of diabetic coma is a cerebral hæmorrhage or thrombosis, occurring in a diabetic. In such cases, starvation and the withholding of insulin may result in glycosuria and ketosis. Both are usually much less severe than in diabetic coma since the patient is often elderly and his diabetes mild. Localising signs, such as hemiplegia, are commonly found but may be absent in subarachnoid hæmorrhage. Beware of missing the fact that a person involved in an accident is a diabetic. In a major catastrophe affecting many people one or two diabetics may be expected.

Treatment.

I. SEVERE KETOSIS

When ketonuria is the only clinical evidence of ketosis, treatment depends upon whether the patient is already on insulin and if so, its type and dosage. As a general rule, any diabetic who shows severe ketonuria should be given insulin and, if the ferric chloride

test is positive, it is wise to start with morning and evening injections of soluble insulin. The dose will depend on age and the severity of the disease as judged by duration, height of the blood sugar, and response to diet. In adults, however, it is usually a waste of time to start with less than 20 to 30 units a day of which rather more than half should be given before breakfast. In untreated cases the effect of an adequate but restricted diet containing between 150 and 200 grammes of carbohydrate should be taken into account when assessing the initial dose of insulin.

In cases on diet and two injections of soluble insulin it may be sufficient to increase both doses but, if this fails to produce a rapid reduction of ketonuria, a third injection of 10 to 20 units, according to the size of the morning dose, should be given before the midday meal until the diabetes is again controlled. If the diabetes was previously controlled by one injection of protamine zinc insulin, it is worth while trying first the effect of adding to it some soluble insulin and testing a specimen of urine passed at about 6 p.m., if this shows heavy glycosuria, a small dose of 10 to 20 units of soluble insulin should be given before the evening meal. Failure to clear the urine of ketone bodies in two or three days by this method, or increase in the degree of ketosis indicates the necessity of stopping the protamine zinc or other delayed action insulin and giving two or three injections of soluble insulin in the manner described.

Diabetics on a mixed dose of soluble and protamine zinc insulin should be treated in the same way, first by the addition of soluble insulin before the evening meal and, if this fails, by the use of three or more injections of soluble insulin. In this group of diabetics, provided that the diet contains an adequate carbohydrate content of not less than 150 and preferably 180 to 200 G. per day, no change need be made, although it is advisable to make sure that the intake of fat does not exceed 100 G. per day.

II. PRE-COMA

Here, a state of actual emergency exists. It is always wise to discontinue delayed action insulins, and rely entirely on multiple injections of soluble insulin. For the treatment of this class of case, it is helpful to have some standard scheme which can be readily understood by the nurse and which can be instituted with the minimum of delay; such a scheme has been worked out

EMERGENCY DIABETIC TREATMENT**Diet and Insulin Control by Urine Tests****DIET**

Give carbohydrate ... grammes. 10 grammes
at carbohydrate
in

Glucose: $\frac{1}{4}$ oz.
Sugar: 2 large lumps.
Orange juice: 4 fl. oz.
Milk: 7 fl. oz.
Benger's: 2 level tea-
spoons.
Horlick's: 2 heaped tea-
spoons.
Ovaltine: 2 very full
teaspoons.

Bread measure ($4 \times 3 \times \frac{3}{4}$ inches) = 10 G. carbohydrate ($\frac{2}{3}$ oz.).

INSULIN

Test urine for sugar before each dose, passing catheter
if necessary.

Given insulin hourly at

IF BENEDICT'S TEST IS :	Marks on Syringe
Red or yellow = + + + sugar.	Give units
Green with copper deposit = Trace of sugar.	Give units
Blue = No sugar.	Give units

TREATMENT CHART

<i>Date</i>	<i>Time</i>	<i>Urine Sugar</i>	<i>Ketones Fe or H</i>	<i>Insulin given</i>	<i>Carbohydrates given as</i>	<i>Remarks</i>
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FIG. 25

The Emergency Sheet.

by Lawrence and is called by him—"The Emergency Sheet" (Fig. 25).

Soluble insulin should be given four-hourly at times recorded on the sheet, with carbohydrate two-hourly in one or more of the suitable forms listed in the top right-hand corner. The dose of insulin will depend on the colour obtained by testing a four-hourly specimen of urine with Benedict's solution, and should be prescribed in the space provided. It is impossible to be dogmatic about doses of insulin, but in diabetics of moderate severity and average insulin sensitivity it may be of the order of 28 to 40 units for a red or yellow test, 12 to 20 for a thick green, and 0 to 8 for a blue; the use of a small dose of insulin in severe diabetics, even when the urine is sugar free, often prevents a serious relapse by the time the next test is made.

If 20 G. of carbohydrate are given every two hours the patient will receive 240 G. in 24 hours, which should be sufficient to cover the insulin and replenish the liver glycogen; later, 40 G. may be given at four-hourly intervals and the insulin dosage modified to allow for the increasing insulin sensitivity associated with diminishing ketosis.

Gastric lavage.

It is a good plan to wash out the stomach with weak sodium bicarbonate solution (one teaspoonful to a pint or 4 G. to 500 ml. approx.) (*see page 537*) before giving anything by mouth.

Dehydration may be a marked feature in pre-coma, especially if this condition has been brought about by continued vomiting. In the absence of repeated vomiting the less severe degrees of dehydration may be corrected by two-hourly fluid feeds alone, or with the addition of water by mouth between feeds. Severe dehydration, with a low or falling blood pressure and decreased ocular tension, to mention only two of the more important signs, should be treated by immediate intravenous saline infusion as in established coma, the rule being: *when in doubt give fluids intravenously*. Much valuable time may be lost and recovery delayed, if not jeopardised, by trying to give fluids orally to severely dehydrated pre-comatose diabetics; such efforts are liable not only to prove ineffectual, but also to cause vomiting and so aggravate both the dehydration and the ketosis.

Treatment of the causal condition.

No less important than the treatment of ketosis and dehydration is that of the underlying cause. Mention of this has been left until the last because, in most cases, such treatment must necessarily follow the institution of the measures already described, while in the comparatively few instances in which this is not so, as for example when the cause is an insulin abscess, treatment is surgical and will be considered under the heading of operations. When infection is present appropriate antibiotics should be used.

III ESTABLISHED COMA

TREATMENT WITH BLOOD SUGAR CONTROL.—Every case of diabetic coma needs constant skilled attention and should therefore be treated in a hospital. Should a diabetic become comatose at home, a difficult decision may have to be made; the more severe and prolonged the coma the greater the need for hospital treatment and the greater also is the risk of moving the patient. It is impossible to lay down hard and fast rules for every case. Obviously the home conditions, availability of doctor and nurses, and proximity of a suitable hospital have a direct bearing on the problem, but perhaps the most decisive single factor is the patient's blood pressure. If the systolic pressure exceeds 100 it is usually best to move the patient immediately; if the figure is much below this level, the possibility of improving the patient's condition before moving him must be seriously considered. The best way of doing this is to give a moderate dose of 40 to 60 units of insulin, and a rapid intravenous infusion of two litres of physiological saline, after which the patient will often be able to stand the journey to hospital without a fatal fall in blood pressure.

Warmth.

On admission the patient should be put into a warmed bed and kept warm with an electric blanket; all other methods are dangerous and less effective, hot water bottles being particularly liable to cause burns unless suitably protected. The foot of the bed should be raised, and blood pressure recorded, the cuff being left in position for subsequent readings at hourly intervals.

Urine and blood sugar.

A catheter specimen of urine should next be taken and examined for sugar, ketone bodies and chlorides, and a sample of blood for sugar estimation.

Intravenous fluid.

All cases of diabetic coma should be given fluid intravenously, preferably into an arm vein without cutting down. The reason for this is that infusion of dextrose solution, in the writer's experience, often causes phlebitis when given into an ankle vein, especially if a cannula is tied in. Blood for sugar estimations is best taken from the lobe of the ear.

Opinions differ as to the best solution for intravenous infusion in the early stages of diabetic coma, but physiological saline (Injection of Sodium Chloride B.P.) is quite satisfactory as it corrects dehydration, hæmo-concentration and chloride loss without the disadvantage, attached to the use of glucose solution, of masking the effect of the initial dose of insulin. The concentration of chloride in physiological saline exceeds that in extra-cellular fluid and tends to accentuate acidosis but if renal function is good this tendency is rapidly corrected. A solution which avoids this tendency contains sodium chloride 5.85 G. and sodium lactate 3.36 G. per litre. If chlorides are completely absent from the urine (Fantus' test, *see page 463*), 2 per cent. saline can be used with advantage until they re-appear but care must be taken not to give too much sodium chloride. Five pints of physiological saline is a safe maximum which should not be exceeded without estimation of the level of chloride in the blood. The first litre should be allowed to run in by gravity as fast as possible and, if dehydration is very severe, a second litre can safely be given in the same way, after which the rate of flow should be cut down to a drip. This should be continued until the patient is well able to take fluids by mouth without vomiting.

Dextrose.

As soon as the blood sugar begins to show a definite fall it is wise to change over to 4 per cent. dextrose in one-fifth physiological saline, and to continue with this solution until the drip is taken down. If the blood pressure continues to fall, the substitution of plasma for saline will sometimes prevent peripheral circulatory failure and is always worth a trial. Dextraven (Benger) 6 per cent. in physiological saline or Plasmosan (May and Baker) may be used for the same purpose. An intravenous noradrenaline drip (Levophed, Bayer) is often effective in maintaining blood pressure. Add 4 ml. of 1 in 1,000 noradrenaline to 1,000 ml. of infusion so that each ml. then contains 4 microgrammes of nor-

adrenaline ; 8 to 20 microgrammes a minute will be needed according to the response.

Potassium and other electrolytes.

While water is taken up comparatively rapidly, electrolytes are only replaced during the first ten days of treatment. Protein restoration begins only after a latent period of some days and probably takes several weeks. Provided that there is a satisfactory urinary flow, it may be assumed that, when the blood sugar level begins to fall, the plasma-potassium level will do the same. In practice, therefore, as soon as hyperglycæmia begins to diminish, intravenous therapy may be continued with a solution containing potassium, phosphate and magnesium. Potassium is the cellular electrolyte most urgently needed by these patients and signs of deficiency (hypokalæmia) may occur during the stage of rehydration. These include severe muscular weakness, shallow respirations leading to respiratory failure, cardiac murmurs, gallop rhythm, tachycardia and a sudden fall in blood pressure. The E.C.G. shows characteristic changes (low T waves, lengthening of the Q.T. interval and depression of the S.T. segment, particularly in lead 2). The plasma potassium level can be quickly estimated by the flame photometer (normal values 20 mg. per 100 ml.) 5 m. Eq/litre. Treatment is as described on *page 466*. Nabarro, Spencer and Stowers advocate the use of Cellular Repair Solution, one litre of which contains:—

Sodium Chloride	1.17 G.
Dibasic Potassium Phosphate	0.87 G.
Potassium Chloride	1.49 G.
Magnesium Chloride	0.24 G.
Glucose	50 G.

This solution should be given not faster than one litre in four hours, and then only if the urinary output is at least 50 ml. an hour.

Insulin.

As soon as possible the patient should be given 60 to 100 units of soluble insulin; if there is marked circulatory collapse, a quarter of the dose should be given intravenously and the rest subcutaneously, using two or more sites in the trunk to obtain rapid absorption. As the initial dose of insulin is almost always given before the level of the blood sugar is known, its size must be in the nature of a guess. The amount suggested

may be insufficient but this error can be easily corrected by a further injection as soon as the blood sugar has been estimated. It should be noted that in an emergency P.Z.I. can be given intravenously since by this route its action is similar to that of soluble insulin.

By the time the drip is set up the initial blood sugar level should be known and the insulin dosage planned accordingly. If the figure exceeds 1,000 mg. per 100 ml., a further 100 units may be given at once; if it is between 750 and 1,000 mg. per 100 ml., 50 units, but if less than 750 mg. per 100 ml., it is probably better to withhold insulin until a second blood sugar level is available. This should be taken three hours after the first so that the level may be known in time to give insulin four hours after the initial injection. The dose of insulin then given will depend on the response to treatment in the first three hours, and the importance of knowing this constitutes one reason for giving saline rather than dextrose intravenously over this critical period. The tendency for the intravenous fluid to lower the blood sugar by dilution should be allowed for in assessing the response of the blood sugar to the insulin already given. If at the end of three hours the blood sugar equals or exceeds the original figure but is less than 1,000 mg. per 100 ml., a further 100 units should be given, 25 units intravenously, 75 units subcutaneously; this dose should be doubled if the blood sugar much exceeds 1,000 mg. per 100 ml. For a poor reduction of 50 mg. per 100 ml. or less, 80 units should be injected, and otherwise 50 units or less according to the blood sugar level. Subsequent dosage can be worked out in the same way from regular blood sugar estimations taken in conjunction with the results of four-hourly tests for sugar and ketone bodies in the urine, a soft rubber catheter being allowed to remain in the bladder for this purpose. As soon as ketosis has been significantly reduced, the patient's sensitivity to the insulin is likely to increase, and the dose of insulin must be correspondingly reduced so as to avoid hypoglycæmia. Lawrence's rule that the dose of insulin in units should be one-tenth of the blood sugar number in milligrammes works well in practice but for blood sugar levels of 500 or less the fasting value of 100 should be subtracted first, *e.g.* blood sugar 400 = insulin dose 30 units. Larger doses of insulin should be given, however, if there is not

an adequate hypoglycæmic response in the early stages of treatment. The later stages of treatment are identical with those described under pre-coma, the Emergency Sheet being used in both cases.

TREATMENT WITHOUT BLOOD SUGAR CONTROL.—No mention has been made of the treatment of diabetic coma without blood sugar control as this should be necessary only in exceptional circumstances. The general principle is the same, but the dosage of insulin must be decided by the results of three or four-hourly urine tests taken in conjunction with the patient's clinical condition. In the early stages, ketonuria is more helpful than glycosuria which will be persistently heavy, the quantitative difference between the ferric chloride and nitroprusside tests explained above being of great importance in estimating the severity of the ketosis and the effectiveness of treatment. The chief difficulty lies in deciding the size of the first few doses of insulin, and one initial blood sugar is of great help in this respect. If this is not available a decision must be based on clinical evidence and the age of the patient. An initial dose of 80 units is suitable for adults in the early stages of coma, 40 units in children and 20 units or less in the very young. Correspondingly larger doses may be needed for coma of longer duration.

As in pre-coma, the draining of local collections of pus, if it involves no major surgery, should be carried out as soon as possible, but major operations should be postponed until the patient is out of coma.

A state of drowsiness may persist for many hours after ketone bodies have disappeared from the urine, and tachycardia may also be present for several days; the latter is probably due to myocardial intoxication and should be treated by complete rest. It has been suggested that the cardiac condition may be the result of vitamin B₁ deficiency and daily injections of 50 to 100 mg. have been advocated for its treatment.

Sudden cardiac failure may occur in the diabetic recovering from coma, particularly if roughly handled. Gentleness in nursing is very important and it is wise not to sit the patient up until the pulse rate returns to normal.

HYPOGLYCAEMIA FROM INJECTED INSULIN

The blood sugar level at which symptoms and signs of hypoglycæmia appear varies in different diabetics, but is

typically not higher than 60 mg. per 100 ml. unless possibly the *rate* of fall is very rapid. There is a time relationship between the onset of hypoglycæmia and type and dosage of insulin injected; the larger the dose, the longer the period during which hypoglycæmia is liable to occur. With soluble insulin in small or moderate doses of up to 30 units, the maximum fall in blood sugar may be expected from three to six hours after injection, but with doses of over 40 units, hypoglycæmia may be delayed for as long as 10 or 12 hours. With protamine zinc insulin the time interval is longer, hypoglycæmia after a morning injection occurring sometimes in the late afternoon, but more often during the night or early morning. Globin insulin resembles protamine zinc insulin but its action is less prolonged, with the result that moderate doses tend to produce their maximum fall in blood sugar about 8 to 12 hours after the injection. In some cases, however, hypoglycæmia may occur before the midday meal.

[It may be mentioned here that although hypoglycæmia occurs most commonly during the treatment of diabetes mellitus, it may also arise in other endocrine disorders such as Addison's disease, Simmonds's disease, pituitary tumours and, rarely, myxœdema. In all these conditions hormones which oppose the action of insulin are deficient. Severe anorexia and vomiting are partly responsible for the hypoglycæmia of an Addisonian crisis, and for the extreme hypoglycæmia in the terminal phase of Simmonds's disease. It has been observed in severe hepatic disease, *e.g.* cirrhosis, where the storage of glycogen is impaired, and in renal glycosuria, where there is abnormal loss of glucose from the body. The secretion of excessive amounts of insulin (hyperinsulinism) results from adenoma, carcinoma or, more rarely, from hyperplasia of the islets of Langerhans]. The symptoms and treatment of hypoglycæmia from whatever cause are the same and are as follows.

Symptoms.

The onset of hypoglycæmia is always rapid, a matter of minutes, or at the most, an hour or so, the patient being in his usual state of health immediately prior to the attack; this fact alone is sufficient to differentiate between hypoglycæmic and diabetic coma in almost every case where an accurate history can be obtained.

The early symptoms resemble the effects of adrenaline and include palpitation, sweating, tremor, restlessness and excit-

ability. There may be intense hunger. Hypoglycæmia, by its action on the central nervous system, may cause mental confusion, inco-ordination, ataxia, paræsthesiæ, especially in the lips and tongue, transient palsies, drowsiness, coma, convulsions, and, rarely, death. To this list may be added nausea and headache which are commoner after overdosage by protamine zinc insulin. Coma is usually preceded by one or more of the early symptoms which, although many and varied, fortunately tend to be stereotyped in individual diabetics. This enables coma to be prevented by the taking of sugar or other carbohydrate in all but the comparatively rare cases in which loss of consciousness occurs almost or completely without warning.

	<i>Hypoglycæmic coma</i>	<i>Diabetic coma</i>
Mode of onset	Sudden	Gradual
Respiration	As in sleep	Deep, abdominal (Air hunger)
Acetone in breath	Absent	Present
Tongue	Moist	Dry and furred
Skin	Moist	Dry
Blood pressure	Normal	Low
Ocular tension	Normal	Low
Ketonuria	Absent or slight	Heavy
Glycosuria	Variable	Heavy
Blood sugar	Less than 60 mg. per cent.	More than 300 mg. per cent.

FIG. 26—Difference between hypoglycæmic and diabetic coma.

Diagnosis.

If the history is not available, diagnosis must be made from the clinical picture and confirmed by response to treatment and, if possible, blood sugar estimation. Treatment should never wait on the result of a blood sugar estimation. In hypoglycæmic coma the skin is pale and moist, and the respiration similar to that of normal sleep; the tongue is moist, the blood pressure and ocular tension normal, and heart rate increased. Signs in the central nervous system may be entirely absent, or there may be evidence of hemiplegia or other palsies with an extensor plantar response on one or both sides. Epileptiform convulsions are not uncommon. The urine contains no ketone bodies but sugar,

although often absent, may be present if the bladder has not been emptied for some time previous to the attack. On the negative side, absence of signs of dehydration and severe ketosis is particularly important in distinguishing between hypoglycæmic and diabetic coma. The main points in the differential diagnosis of these two states are summarised in Fig. 26.

Treatment.

PROPHYLACTIC.—Frequent attacks of hypoglycæmia mean bad diabetic treatment. They often result from an attempt to render the urine sugar free after meals instead of before them. The use of buffer feeds in the mid-morning and at bed-time will often prevent attacks, and a late feed should always be given to patients on protamine zinc insulin to reduce the risk of nocturnal hypoglycæmia.

THE TREATMENT OF THE ATTACK.—This consists in giving the diabetic, or more often in his taking, glucose or some other rapidly absorbed form of carbohydrate such as sugar, jam, biscuit, bread, or chocolate in adequate amounts when symptoms begin. There is a general tendency on the part of diabetics and those treating them to give too little carbohydrate, especially in attacks due to protamine zinc insulin, with the result that either the attack is not relieved or recurs after a brief interval of improvement. Four lumps of sugar or their equivalent (20 G. of carbohydrate) is a reasonable initial dose, and should be repeated in 15 minutes if symptoms persist.

With the onset of coma, treatment at once becomes more urgent and less simple, as carbohydrate food can no longer be swallowed. This difficulty can be overcome in a number of ways, the best of which is to give concentrated dextrose solution intravenously; a 20 ml. syringe of a 60 per cent. solution (= 12 G.) is sufficient to bring all but the most severe cases out of coma in a few minutes. If sterile dextrose solution is not available and coma is not too deep 0.6 ml. (10 minims) of Injection of Adrenaline B.P. subcutaneously may revive the patient sufficiently for sugar to be given by mouth. A solution of glucose or sugar may be given by means of a stomach tube or nasal catheter, the former being the safer method for those not experienced in the art of nasal feeding. Rectal glucose is inefficient in raising the blood sugar and should not be relied upon in the treatment of hypoglycæmic coma. It is scarcely necessary to add that though sucrose

is effective by mouth, it is useless to give it intravenously or by rectum.

When consciousness has been regained the patient should be given a carbohydrate feed and carefully watched for evidence of relapse.

A small number of cases of profound and prolonged coma fail to respond to elevation of the blood sugar and either die or only slowly regain consciousness after a period of many hours or even days; such cases are liable to exhibit temporary or permanent mental and nervous changes from damage to the brain.

Whenever there is the least doubt about the cause of coma in a diabetic it is a safe rule always, first to give dextrose without insulin and, if coma is due to hypoglycæmia rapid recovery is likely to result; if it is diabetic, no harm will be done.

OPERATIONS

Minor operations.

If only a local or brief general anæsthetic such as gas and oxygen, or thiopentone (Pentothal) is required, operation can be carried out with little disturbance to the diabetic régime. The usual insulin and diet should be taken up to the last meal before the anæsthetic and this should consist only of the carbohydrate portion. Sufficient time should be allowed after this meal for the stomach to empty and nothing should be given by mouth for three hours before operation.

Major operations.

While almost any anæsthetic except chloroform may now be given to a diabetic, providing that the diabetes is reasonably well controlled and the patient suitably prepared, the shorter the period of unconsciousness following the operation, the smaller will be the risk of post-operative ketosis. Anæsthetics such as ether, however, which tend to cause post-operative vomiting, are best avoided. When severe ketosis is already present, unless immediate operation is necessary to save life, it is wise to treat the patient according to the Emergency Sheet (*page 236*) until the ketosis has cleared up, before an anæsthetic is given; by so doing the risk of diabetic coma following the operation is greatly diminished. When operation is not urgent, the patient should first be stabilised on two or more injections of soluble insulin and kept on it until the usual diet is resumed.

On the day of operation, the procedure will depend on the time the operation is to take place. The general principle is to give enough carbohydrate and insulin before operation to build up an adequate supply of liver glycogen, and at the same time send the patient to the theatre with an empty stomach and not too high nor too low a blood sugar. Nothing should be given by mouth for at least four hours before the induction of an anæsthetic. In cases of emergency in which there is no time for careful preparation it is always wise to pass a tube and make sure that the stomach is empty before the anæsthetic is administered. When there is no urgency the patient should receive rather less than his usual morning dose of soluble insulin together with his full amount of breakfast carbohydrate; four hours before operation an additional 50 G. dextrose should be given as a well-diluted and preferably iced drink. When operation is timed for noon or earlier this drink may be substituted for the breakfast carbohydrate. For operations in the later part of the day the normal routine should be observed until four hours before the anæsthetic when the patient should receive rather less than his usual evening dose of soluble insulin together with the 50 G. glucose drink. When large doses of insulin are being used or it is thought that the four-hour fast may result in hypoglycæmia during or shortly after operation, it is a good plan to give 15 to 20 G. dextrose intravenously as soon as the patient is under the anæsthetic.

In all major operations sterile dextrose should be available for intravenous injection in case the patient vomits before or during the administration of the anæsthetic. Post-operatively a pint of 5 per cent. dextrose may be given slowly into the rectum, but whenever the patient is unable to take fluids by mouth for more than a few hours after operation dextrose should be given as a drip either intravenously or into the stomach.

WILFRID OAKLEY.

CHAPTER XIV

Medical Emergencies in Other Endocrine Disorders

THE THYROID GLAND

Toxic goitre.

THE emergencies which may arise in a patient with toxic goitre are the thyroid crisis and congestive heart failure, which may or may not be associated with a crisis.

(a) Thyroid crisis.

This condition may occur either during the course of the disease or after operation, and is characterised by a rapid increase in the symptoms of thyrotoxicosis. The patient becomes restless and irritable, is sweating and flushed, and often delirious. The pulse rate may rise to 200 or more and there is a rise of blood-pressure and temperature, which may be as high as 106°F. Pulmonary oedema may be present. Later the patient may become collapsed with a pale, cool, clammy skin, and death is the result in most cases, often within a few hours of the onset.

The mechanism of a crisis is obscure. It may be precipitated by psychological disturbances. It has been suggested that an excessive secretion of adrenaline is responsible, since the symptoms are comparable to those of an overdose, but there are probably other factors. Since the fever is not due to infection, there is evidently a breakdown in the heat-regulating mechanism. One hypothesis that has been suggested is that the condition is caused by acute thyroid failure, but the syndrome certainly does not occur in myxoedema.

PREVENTION.—In the management of a severe case of toxic goitre, all precautions should be taken to prevent a crisis, because once it occurs, the outlook is grave. Rest in bed, the avoidance of psychological upsets, a high calorie diet with abundant carbohydrate and the administration of a sedative, such as phenobarbitone 30 mg. (gr. $\frac{1}{2}$) three times a day, are important. Operation on such a patient should never be "rushed," but should only be undertaken when the maximal improvement has been obtained with medical treatment. The incidence of crisis fell considerably

as a result of the introduction of the pre-operative use of iodine. Now, by means of methylthiouracil, severe cases can be got into a still more satisfactory state for subtotal thyroidectomy, so that the two-stage operation, which used to be employed to minimise the surgical risk, is rarely necessary. In severe cases methylthiouracil is given as a pre-operative measure in doses of 100 mg. three times daily until the maximal improvement is obtained, usually in four to eight weeks. As methylthiouracil produces a vascular and friable thyroid gland which renders operations difficult, the drug is stopped when its full effect has been obtained and Lugol's solution (Aqueous Solution of Iodine B.P.) is given in doses of 0.3 ml. (m 5) three times daily for two to three weeks before the operation. The effect of methylthiouracil is maintained for about four weeks and the iodine reduces the vascularity of the gland. Throughout treatment the winning of the confidence and co-operation of the patient is essential.

In severely toxic patients with diffuse goitre Lugol's solution 0.6 ml. (m 10) three times a day may be necessary for ten days at the beginning of treatment in conjunction with methylthiouracil in order to bring about a more rapid improvement than can be effected by thiouracil alone. It should be resumed during the two or three weeks before operation as described below.

Treatment.—The importance of the correct management of toxic goitre has been stressed because it is so great a factor in preventing a crisis. Should this occur, a large dose, 4 ml. (m 60), of Lugol's Solution (Aqueous Solution of Iodine B.P.) in milk should be given at once, followed six hours later by 2 ml. (m 30), which should be repeated six-hourly for the next 24 hours. If there is much restlessness and mental disturbance (and this is usually the case), phenobarbitone 180 mg. (gr. 3) should be given by mouth, or morphine 10 mg. (gr. $\frac{1}{6}$) subcutaneously. The patient should be kept cool with ice packs, and the early and continuous administration of oxygen is usually beneficial. Since full stores of glycogen in the liver are said to reduce the incidence of crises, 500 ml. of 10 per cent. dextrose in Sterilised Water for Intravenous Injection B.P. may be given intravenously and be repeated at intervals. Some authors recommend the addition of 0.3 to 0.6 G. (gr. 3 to 10) of sodium iodide to the intravenous dextrose solution.

(b) Congestive heart failure.

This occurs as a result of the added burden of thyrotoxicosis on an already damaged myocardium. Failure may or may not be associated with auricular fibrillation. Acute congestive failure in toxic goitre should be treated on the same lines as congestive failure unconnected with thyrotoxicosis, but it should be remembered that the thyrotoxic patient has a greater sensitivity to drugs such as morphine, which should therefore be given in slightly smaller dosage. If failure is immediately endangering the life of the patient, Lugol's solution should be given at once in order to diminish the degree of thyroid intoxication, instead of postponing it until the full effect of digitalis has been obtained. During the first 24 hours 1·8 ml. (m 30) should be given six-hourly, and thereafter 0·6 ml. (m 10) three times a day.

(c) Acute thyrotoxic myopathy. (Acute thyrotoxic bulbar palsy—Laurent.)

This is a very rare condition which may occur in severe thyrotoxicosis. It is a rapidly spreading bulbar palsy with paralysis of the muscles of mastication and deglutition, facial weakness, ptosis, diplopia, paresis of the muscles of the neck and generalised weakness of the limbs.

It is practically always fatal within one or two weeks of onset. Prompt relief of the paralysis occurs after the subcutaneous injection of neostigmine (Prostigmin), and in this the condition resembles myasthenia gravis.

J. H. Sheldon and R. Milnes Walker* have described a case in which treatment with Prostigmin and partial thyroidectomy was followed by rapid recovery. After the initial subcutaneous injection of 1 mg. of neostigmine (Prostigmin), the drug was given by mouth in doses of 15 mg., four times a day. After the partial thyroidectomy, it was continued in doses of 90 mg. daily by mouth, and gradually reduced.

Substernal goitre.

Unless it is substernal, simple goitre does not endanger life. Long-standing nodular goitres may rapidly increase in size through sudden hæmorrhage into a cyst. If this occurs in a substernal goitre, sudden pressure on the trachea may cause asphyxia, coma, and death. On examination, part of the enlarged gland may or may not be palpable above the sternum, but there is always dulness on percussion over the manubrium. In less urgent cases an X-ray examination should be made, and will reveal a substernal mass and deviation of the trachea. Treatment consists in immediate partial thyroidectomy.

**Lancet*, 1946, 1, 342.

THE PARATHYROID GLANDS *

Tetany is the name given to a state of increased neuromuscular excitability consequent upon a reduction in the concentration of *ionised* calcium in the tissues. The condition may be associated with a variety of diseases, but the clinical picture is essentially the same in them all—muscular twitchings (especially on percussion), carpo-pedal spasm, drowsiness, stridor and, in severe cases, even convulsions and coma.

The underlying causes may be placed in two groups. Firstly, those represented by rickets, osteomalacia, coeliac disease, idiopathic steatorrhœa, renal dysfunction and hypoparathyroidism either following operation or occurring spontaneously; in all of these there is a diminution in the total serum calcium and a proportionate fall in the amount of "available" or ionised calcium. Secondly, there are cases in which the total calcium in the serum is normal, but on account of an alkalotic state in the tissues, too little of it has ionised and become "available" and thus patients suffering from persistent vomiting (loss of chloride), hyperventilation with excessive depletion of volatile acid (carbon dioxide) as in hysterical hyperpnœa, and prolonged overdosage with easily diffusible alkali (*e.g.*, bicarbonate of soda) fall into this group.

The emergency treatment of tetany, when it is associated with low serum calcium, is to raise the level of calcium by parenteral injection. In urgent cases, *i.e.* those with painful spasm of the limbs and severe laryngeal spasm, calcium gluconate, 10 to 20 ml. of a 20 per cent. solution should be *slowly* injected intravenously. In less urgent cases with mild spasm of the hands and feet only, it may be given intramuscularly. Calcium chloride 20 to 40 ml. of a 5 per cent. solution, may also be given intravenously, but it has the disadvantage that if any escapes into the subcutaneous tissues it may cause necrosis and ulceration. For this reason it should never be given intramuscularly. With the parenteral administration of calcium, the spasms rapidly subside. Shortly after the intravenous injection, it is as well to give an intramuscular injection of the same amount of calcium gluconate, so that with its slower absorption the effect is prolonged. Thereafter the patient receives treatment for the chronic state, and for the underlying cause. If the spasms recur, the injections should be repeated. In very severe convulsions making intravenous injection difficult, sedatives such as chloral hydrate 1·8 G. (gr. 30) by

mouth or paraldehyde rectally (*see page 597*) may be given. Inhalations of chloroform might be used. These are only temporary measures to quieten the patient and should not take the place of the administration of calcium.

In parathyroid tetany, which may arise 24 hours to one week after thyroidectomy, the same treatment is adopted but in addition 20 to 30 units of a parathyroid extract (*see page 655*) should be given intramuscularly, 6 to 8 hourly. It acts by withdrawing the calcium from the bones, thus raising the level of calcium in the blood, but since it takes from 4 to 6 hours to achieve this, it would not be effective in an emergency. In severe cases the first dose should be 50 to 60 units given intravenously. When parathyroid extract is used, symptoms of hypercalcaemia—drowsiness increasing later to coma—may occur. Treatment consists in stopping the injections and reducing the increased viscosity of the blood caused by the excess of calcium, by withdrawing a pint of blood and injecting one litre of physiological saline intravenously. Parathyroid extract is not used in the treatment of chronic parathyroid tetany because it loses its effectiveness after a few weeks. At this stage, however, tetany can be kept in abeyance by giving calcium and vitamin D by mouth.

The essential treatment of tetany caused by alkalosis is to adopt appropriate measures to counteract the alkalosis, and thereby increase the ionisation of calcium. Alkalosis caused by repeated vomiting is produced by excessive loss of hydrochloric acid and chloride in the vomitus, and is treated by supplying chloride in the form of physiological saline intravenously, and quarter strength physiological saline by mouth. When tetany arises through giving alkali, the drug should be stopped; and if this is not effective, a few doses of an acid-forming salt, such as ammonium chloride, 0.6 to 0.9 G. (gr. 10 to 15), may be given, but this is rarely necessary. In tetany caused by overbreathing, the alkalosis is produced by the washing out of carbon dioxide from the tissues and the acute attack is treated by giving an inhalation of 7 per cent. carbon dioxide in oxygen. If this is not obtainable the patient's expired CO₂ may be used by letting him re-breathe from a bag containing air or oxygen. The underlying hysteria should subsequently be treated.

Acute hyperparathyroidism.

This is an extremely rare condition, caused by an adenoma of one of the parathyroid glands. Its diagnosis may be easily overlooked. The

principal features of the disease are anorexia, vomiting, abdominal pains and constipation, lassitude, asthenia, pains in the bones and loss of weight, slight fever with disproportionate tachycardia, evidence of impaired renal function without hypertension and, terminally, increasing drowsiness. The serum calcium is greatly raised and there is widespread visceral and vascular calcification. If untreated the disease is rapidly fatal.

If the condition is recognised early and treated promptly, good results may be expected. The emergency treatment consists in endeavouring to lower the viscosity and the calcium level of the blood by withdrawing a pint or more of blood from a vein and replacing it by twice the volume of physiological saline. This procedure is followed by removal of the parathyroid adenoma.

THE ADRENAL GLANDS

Crisis of Addison's disease.

Patients with Addison's disease are liable to crises which are precipitated by cold, exertion, infection and operation, or by an extension of the disease process. The early symptoms are increasing weakness, vomiting, diarrhoea, mental changes, and a fall in temperature and blood pressure. Sometimes a "peritonitis-like" syndrome develops in the terminal stages of Addison's disease. The serum sodium and chloride are usually low (normally 330 mg. per 100 ml. [144 mEq/litre] and 585 mg. per 100 ml. [100 mEq/litre] respectively), and the blood urea and non-protein nitrogen practically always high (normally 20 to 40 mg. per 100 ml.). There is usually, but not always, hypoglycæmia. If untreated the patient develops profound exhaustion, dehydration and collapse, and dies in coma. It is important to treat these cases early, for if treatment is delayed the patient sinks into a refractory state which is unresponsive to therapy.

In a known case of Addison's disease the diagnosis of an impending crisis presents no difficulty, and should be suspected if rapidly increasing weakness, vomiting or diarrhoea occurs. In rare cases a crisis may be the presenting feature. If pigmentation of the skin is absent or slight, or the buccal patches are overlooked, the condition may then be mistaken for a severe gastrointestinal infection. The finding of a low blood sodium is valuable in confirming the diagnosis and this estimation should be repeated at intervals in order to assess the results of treatment.

Treatment.—Especially if there is fever search should be made for infection and appropriate antibiotic treatment started. While the fundamental cause of a crisis is acute adrenocortical insufficiency, the precipitating factor may be dehydration or hypoglycæmia or both. In addition to giving cortical hormones it is important to

combat these two conditions. After taking a sample of blood for analysis and without waiting for the results give 50 ml. of 50 per cent. dextrose intravenously. At the same time inject 10 mg. of Depoxycortone Acetate B.P. in oil intramuscularly for a slow continuous action over 24 hours and give hydrocortisone intravenously. A 20 ml. vial contains 100 mg. of hydrocortisone free alcohol in 50 per cent. alcohol; before injection it should be diluted to at least 500 ml. with physiological saline and given by continuous drip over a period of six hours. If the response is unsatisfactory the rate and duration of flow should be increased. Intravenous hydrocortisone is rapidly excreted and so when about half the infusion has been given 50 mg. of cortisone acetate should be injected intramuscularly so that there may still be corticoid action. Six hours after the initial dose 50 mg. of cortisone acetate should be injected and this dose continued six-hourly for the next 24 hours. When the infusion of hydrocortisone is completed about two litres of 5 per cent. dextrose in physiological saline should be given during the next 12 to 24 hours but waterlogging must be avoided.

Hydrocortisone is given intravenously for a rapid effect because, although oral cortisone is rapidly absorbed, vomiting or drowsiness may preclude its administration. Epileptiform convulsions can be precipitated in susceptible patients by intravenous hydrocortisone but their occurrence is rare. Deoxycortone is necessary also because it is the most active hormone in the maintenance of life and is much more potent than cortisone and hydrocortisone in causing sodium retention.

During the second day the patient should be given milk, chocolate and fruit juices. An intranasal tube may be necessary. Further treatment must be regulated by the clinical state and the biochemical findings. If there is still evidence of dehydration, hypotension, hæmoconcentration and urea retention a further 1 to 2 litres of physiological saline will be necessary containing, if hypoglycæmia is present, 5 per cent. of dextrose. Cortisone acetate 50 mg. six to eight-hourly and deoxycortone acetate 5 mg. in oil every 24 hours should be given.

On the third day 5 mg. of deoxycortone acetate should be given intramuscularly and intravenous dextrose saline should be repeated if the patient cannot tolerate fluids by mouth. It may be possible to reduce the cortisone injections to 12-hourly intervals.

On the fourth day Tab. Cortisone Acetate 25 mg. six-hourly may be tolerated by mouth and 5 mg. of deoxycortone acetate should be repeated intramuscularly. Salt by mouth in three 1 G. doses in gelatin capsules or dissolved in fruit juice should be started but if it is not tolerated a 1 per cent. solution should be given intravenously. No more than 3 G. of added salt a day should be given when the patient is receiving deoxycortone acetate lest it causes oedema.

On the fifth day the patient should be able to take a more solid diet, which should be rich in carbohydrates. The administration of cortisone and deoxycortone should be continued in the same dosage, but thereafter cortisone should be gradually reduced to the maintenance dose of 12·5 to 37·5 mg. daily and that of deoxycortone to 1 to 2 mg. daily.

If hydrocortisone and cortisone are not immediately available, recourse must be had to adrenal cortical extract. At the beginning of treatment 50 ml. of Injection of Suprarenal Cortex B.P.C. should be given intravenously (it acts immediately) and 20 ml. intramuscularly (this has its maximum effect in three to six hours), together with 10 mg. of Deoxycortone Acetate B.P. in oil intramuscularly. Cortical extract 20 ml. should be repeated intramuscularly four- or six-hourly during the first 24 hours. On the second day give 5 to 10 ml. of cortical extract intramuscularly four- to six-hourly and one intramuscular injection of 10 to 15 mg. of deoxycortone acetate in oil. On the third day give two injections of extract and 10 mg. of deoxycortone acetate. On the fourth day cortical extract may be discontinued if the patient's condition warrants it, but 10 mg. of deoxycortone acetate should be repeated. On the fifth day the dose of deoxycortone acetate should be reduced to 5 mg. daily and thereafter treatment is continued as for the chronic case.

Instead of cortical extract an aqueous solution of deoxycortone glucoside (Ciba) may be injected intravenously in doses of 50 mg. once or twice daily until the acute phase has passed; it should be supplemented by intramuscular injections of deoxycortone acetate in oil in doses of 5 mg. six-hourly.

Surgical complications in Addison's disease. (*see also page 68*)

Patients with Addison's disease stand surgery badly and when a surgical emergency arises, especially if abdominal, adequate pre-operative treatment is necessary. If adrenal deficiency is not

completely controlled and operation urgent, it is wiser to pay attention to pre-operative preparation rather than to risk immediate surgery.

On the day before operation 10 mg. of deoxycortone acetate should be injected intramuscularly and, at six-hourly intervals, 50 mg. of cortisone acetate. One hour before the operation a further 5 mg. of deoxycortone acetate and 100 mg. of cortisone acetate should be given. Five per cent. dextrose in 1·5 per cent. saline should be injected by intravenous drip during the operation and for 24 hours afterwards. After the operation 50 mg. of cortisone acetate should be injected intramuscularly six-hourly during the first 24 hours and twelve-hourly during the second 24 hours, and on the day after the operation one injection of 5 mg. of deoxycortone acetate should be given.

If cortisone is not available, the following procedure should be adopted. 10 to 20 ml. of Injection of Suprarenal Cortex B.P.C. (cortical extract) should be given intramuscularly at eight-hourly intervals on the day before the operation and one intramuscular injection of 10 mg. of deoxycortone acetate. One hour before the operation 25 ml. of cortical extract should be injected intravenously and 10 mg. of deoxycortone acetate intramuscularly. After the operation a further 25 ml. of cortical extract should be injected intramuscularly. On the day after the operation 10 ml. of cortical extract should be injected intramuscularly eight-hourly and 5 to 10 mg. of deoxycortone acetate. The dextrose-saline solution should be given during and after the operation as described in the preceding paragraph.

Local anæsthesia should be employed when possible, and failing that, nitrous oxide, oxygen and ether. It should be remembered that patients with this disease are sensitive to morphine and narcotic drugs.

Pregnancy in Addison's disease.

Labour with its physical stress and post-partum stage, when the foetal cortical hormones are no longer available, are serious and may precipitate a crisis. The patient should be prepared for labour as for a surgical operation (*see above*); this treatment should be continued until the danger of a crisis has passed.

Other forms of acute adrenal insufficiency.—Acute adrenal insufficiency may be caused by acute infections, thrombosis or hæmorrhage in the adrenal glands (Waterhouse-Friderichsen syn-

drome); it may thus complicate bleeding diseases such as purpura hæmorrhagica. Its features are similar to those of an Addisonian crisis, consisting of extreme asthenia and prostration, vomiting, diarrhoea, abdominal pain, dehydration, low blood pressure and coma. It may be accompanied by fever, and in cases of adrenal hæmorrhage there will be local signs of abdominal distension and perhaps a palpable swelling in one or both kidney regions.

Acute adrenal insufficiency may arise after bilateral adrenalectomy as a result of an infection or some other form of stress. The clinical features are somewhat atypical: the patient feels ill and weak and his condition deteriorates rapidly; nausea and vomiting are common and the blood pressure falls. These forms of adrenal insufficiency should be treated on the same lines as an Addisonian crisis.

It would be pertinent to draw attention to the fact that patients with intact adrenal glands who are receiving cortisone therapy for other diseases, develop adrenocortical atrophy through suppression of the secretion of corticotrophin (A.C.T.H.) by cortisone. Should these patients acquire an infection or be subjected to a major operation, they may show adrenal insufficiency. The dose of cortisone should then be increased and before and during the operation hydrocortisone should be administered by intravenous drip.

Acute adrenal insufficiency secondary to hypopituitarism is discussed on *page 259*.

Paralysis due to overdosage by deoxycortone acetate.

Excessive dosage of deoxycortone acetate in Addison's disease, or in conditions erroneously diagnosed as such, may result in a profound fall in the serum potassium. This may be associated with widespread muscular paresis or paralysis, the condition being somewhat similar to familial periodic paralysis, which is also accompanied by a low serum potassium. In a case seen by the author, although the serum potassium value was very low, the levels of serum sodium and serum chloride were normal, suggesting that deoxycortone has a specific effect on potassium metabolism.

The paralysis or muscular weakness rapidly disappears as a result of stopping the administration of deoxycortone acetate and giving potassium salts. In the case mentioned 12 G of potassium chloride were given in divided doses throughout the day; the paralysis, which affected all four limbs, cleared up in 36 hours.

Adrenal medullary tumour: Phæochromocytoma (hyperchromafinism).

This rare tumour, which is usually non-malignant, secretes adrenaline and noradrenaline, and may cause paroxysmal or continuous hypertension. A hypertensive attack or crisis consists of

sudden dyspnoea, tachycardia, headache, pallor, "goose flesh," a feeling of constriction about the heart and epigastrium, and sometimes nausea and vomiting. There may be glycosuria, and pulmonary oedema may occur. Sweating is usually a prominent symptom. Coma may result from hypertensive encephalopathy, and in some instances sudden death has been reported. A tumour may be palpable in the renal region, but if not, the case may be mistaken for one of hypertensive encephalopathy complicating essential hypertension. Pyelography may reveal displacement of the kidney by the tumour and enlargement of the adrenal gland may be seen in X-ray films taken after peri-renal insufflation of oxygen (which is not without risk of air embolism, *see page 26*) or by tomography.

The emergency treatment for the condition is as for hypertensive encephalopathy, *i.e.*, rest, a venesection of 568 ml. (20 fl. oz.) of blood (*page 580*) and a lumbar puncture (*page 522*) to relieve the increased intra-cranial pressure.

When the tumour is being removed adrenaline and noradrenaline may be discharged into the circulation as a result of emotional stress, anaesthesia or surgical manipulation. One of the adrenergic blocking agents (benzodioxane, dibenamine, phentolamine) should be employed to prevent any paroxysms of hypertension. These drugs decrease or abolish hypertension when it is caused by excess of adrenaline and noradrenaline and are believed to act by combining with the adrenaline-specific receptor, thereby competing with adrenaline.

The most satisfactory preparation is phentolamine ("Rogitine") which is remarkably free from significant side-effects. A few minutes before operation 5 mg. of phentolamine are injected intravenously and during the operation the same dose should be administered by the same route whenever the blood pressure begins to rise as a result of manipulation (Fig. 27). The injection of phentolamine may be repeated if necessary to maintain a reasonable level of blood pressure. Should the systolic blood pressure fall below 100 mm. of mercury 0.3 ml. (5 minims) of 1 in 1,000 adrenaline hydrochloride solution should be injected subcutaneously at half-hourly intervals for the next 12 to 24 hours, or 1 ml. of the adrenaline solution should be added to every 500 ml. of isotonic saline administered intravenously by continuous drip.

Further adrenaline may be necessary during the subsequent 24 hours.

It has been suggested that the "shock," collapse and fall of blood pressure that may occur after removal of the tumour may also be due to deficiency of adrenal cortical hormones. In order to prevent collapse of this nature the patient should be given deoxycortone acetate in doses of 5 mg. intramuscularly on the day preceding, just before and immediately after the operation; this dose should be repeated daily for two or three days.

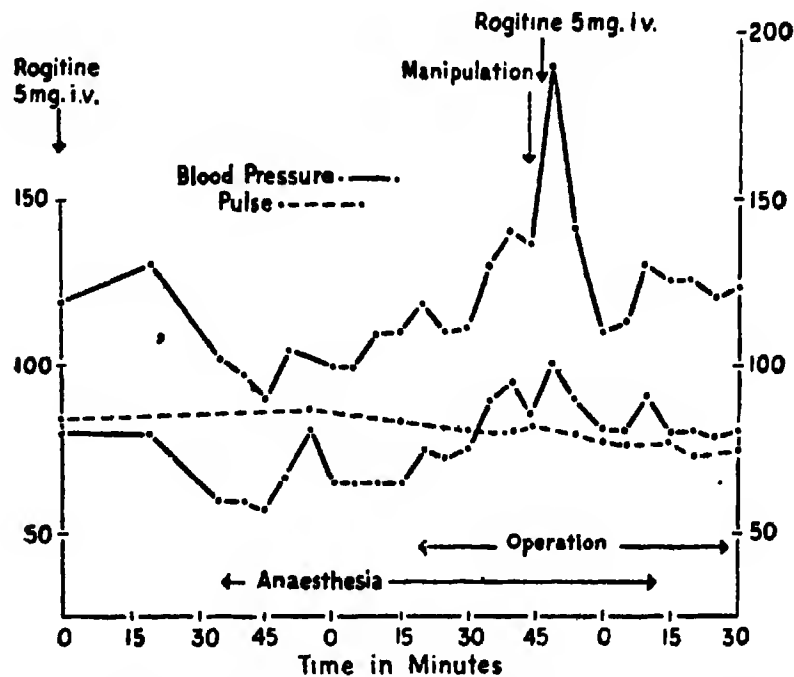


FIG. 27—Demonstrating the effect of phentolamine ("Rogitine") on the blood pressure during removal of a pheochromocytoma. (Zintel, 1952.)

THE PITUITARY GLAND

Hypopituitary coma.

Hypopituitary coma may develop in patients with Simmonds's disease, which is caused by any lesion which damages the anterior lobe of the pituitary gland. The commonest one is post-partum necrosis due to thrombosis in the artery supplying the anterior lobe, precipitated by hæmorrhage and collapse at childbirth. The precomatose state is characterized by rapid deterioration in the patient's condition, manifested by drowsiness, twitchings or convulsions, a fall of blood pressure, a poor pulse volume and shallow respirations. The patient may later sink into coma. The precipi-

tating factors are operation on a pituitary tumour, infections, hypoglycæmia, sodium depletion, water retention, cerebral anoxia, hypothermia and the administration of drugs and anæsthetics to which the patient is sensitive. The fundamental cause is acute failure of adrenocortical function.

The treatment of hypopituitary coma is the same as that of an Addisonian crisis (*see page 253*). In cases of hypothermia, however, the most promising treatment is to raise the body temperature by immersing the patient in a hot bath.

Hypoglycæmia in endocrine disease is described with hypoglycæmia from injected insulin on *page 242*.

ALLAN W. SPENCE.

CHAPTER XV

Medical Emergencies in Renal Disease

ACUTE RENAL FAILURE

(Acute tubular necrosis; anuria)

THE composition of the body fluids in health is remarkably constant. Though we take little heed of the amount of water, salt and protein we ingest, the kidneys, partly under orders from the pituitary, parathyroid and adrenal glands, faithfully, accurately and promptly discard the excess, be it water, salt, potassium, phosphate, calcium, nitrogen, acid or alkali.

The greatest of all renal emergencies is therefore failure of the kidneys to perform this regulatory function, that is, when they secrete little or no urine. Then even water may become a poison, salt may cause pulmonary oedema and excessive potassium intake (a fruit diet for instance) may cause fatal paralysis and cardiac standstill.

The principles of conservative treatment are clear: since no electrolytes are being excreted none must be taken in, and the same would apply to water but for the fact that some will be lost from the skin and in the expired air, and this must be replaced. Protein intake must cease, and the natural katabolism of body proteins must be reduced to a minimum by providing adequate calories from carbohydrate or fat. Having discussed principles we may now turn to diagnosis and treatment.

Acute renal failure occurs in a number of conditions. Most commonly it follows traumatic or hæmorrhagic shock when the patient has been in a state of circulatory collapse and hypotension for some hours. During this time the renal circulation has probably been largely shut off and renal tubular necrosis results. Well recognized causes are: severe trauma, including the crush syndrome, anaphylaxis, concealed accidental hæmorrhage, abortion, and post-partum hæmorrhage. Mis-matched blood transfusion, blackwater fever, and mercurial salts may also cause tubular necrosis. Anuria may also occur in acute nephritis (*page 268*).

After the patient has been resuscitated from shock, usually by blood transfusion, it becomes noticeable that he is passing very

little urine. Complete anuria shortly develops, or at most the daily urine is only a few ounces. It is now well established that if the patient can be kept alive long enough, the secretion of urine may return, sometimes after as long as 15 or 20 days. It is therefore most important that the correct principles of treatment be put into action from the beginning. Only if there is clear evidence of dehydration, and a history of fluid loss from vomiting or diarrhoea should intravenous saline or other fluid be given (except in the special case of feeding via the vena cava as described below). If the blood volume is judged to be still low, as a result of hæmorrhage, adequate blood transfusion should be ensured.

Conservative treatment must then be instituted. A diet consisting solely of carbohydrates and fat without electrolytes or protein is unpalatable and liable to cause vomiting, which is not merely a discomfort but a danger to the patient. Nevertheless Oard and Walker (*Amer. J med.*, 1955, 18, 199) have used oral feeding consisting of 500 ml. of 20 per cent. lactose daily, the amount of water being increased if there is thirst or dehydration. These authors ignore a fall in serum chloride and bicarbonate which commonly occurs and state that on this regime serious hyperkalæmia rarely occurs in recoverable cases. The second method is to feed the patient by intra-gastric drip. The solution originally recommended by Bull and his colleagues consisted of glucose 400 G., peanut oil 100 G., with some acacia to emulsify, and water to 1 litre. Any fluid vomited was filtered and put back into the solution, to replace loss of electrolyte. Bull now states that the peanut oil sometimes gives rise to diarrhoea and that the carbohydrate is the more important part of the regime. The third alternative is to give a highly concentrated solution of carbohydrate through a polythene catheter passed up the saphenous and femoral veins into the inferior vena cava. The rationale of this method is that because of the limited permissible fluid intake and the importance of supplying calories, the carbohydrate solution necessary for feeding is so concentrated that it will rapidly cause thrombosis if put into a peripheral vein. This method may be life-saving in cases where the intragastric drip is not tolerated or causes vomiting or diarrhoea. Chalmers and Fawns used 40 per cent. dextrose to which 15,000 units of heparin were added daily. They gave sodium lactate (M/6 solution) in addition when the plasma bicarbonate showed that severe acidosis had developed.

There is no doubt at all that these conservative measures have saved many lives and that many patients were killed in the past by misdirected and unsuccessful efforts to force the kidneys to secrete. Chief amongst these misdirected efforts was the administration of intravenous sodium sulphate.

The question now is whether anything else is necessary besides the conservative regime. Units equipped with special methods for extra-corporeal dialysis of plasma (the artificial kidney) will not need the aid of a manual such as this, but it is probably fair to say that these methods are still in the experimental stage and carry their own special risks.

Cases being treated conservatively should preferably be in close touch with laboratory services so that frequent (for instance daily) samples of blood can be analysed for sodium, potassium, chloride, bicarbonate and urea. The blood urea will of course steadily rise and may continue to rise for two or three days after diuresis has set in. Blood ureas as high as 500 mg./100 ml. are still compatible with survival. A certain amount of hæmodilution as shown by low values for sodium and chloride accompanied by symptoms of water intoxication (cramps, mental confusion or convulsions) should be treated cautiously by the intravenous injection of about 200 ml. of 5 per cent. NaCl, or molar sodium lactate if acidosis coexists. The best rule however is not to treat serum chemistry unless symptoms exist which can be attributed to its disturbance. The one possible exception is where serious excess of potassium is developing (over 8 mEq per litre). This is more likely to occur in cases of trauma where muscle necrosis releases potassium. Before treating this emergency the estimation should always be repeated on a fresh sample of serum however, because falsely high readings often result when serum has been left in contact with red cells, or when hæmolysis has occurred. A high serum potassium can be corrected by encouraging potassium to shift from extracellular fluid to cells, by giving glucose and insulin, but this is not very effective where hypertonic glucose is already being given. Resins are not well tolerated, but a resin in the sodium cycle (*e.g.* Resonium A, 10 G. three times a day by mouth) can be tried. Calcium gluconate (10 ml. of 10 per cent. solution intravenously) may counteract the ill-effects of hyperpotassæmia. Exchange transfusion (*see page 592*) has occasionally been used with good effect. Otherwise, some form of dialysis may be necessary.

Hyperpotassæmia should not usually develop if the case is properly treated from the start and the carbohydrate intake has been sufficiently high. Dialysis, assuming no "artificial kidney" is available, can be done by peritoneal lavage, glucose or saline solution being run into the peritoneum through a cannula and allowed to drain off later. The procedure carries the hazard of infection; fibrinous deposits form around the indwelling tube, and the method is not generally to be recommended. (*For details of technique see Grollman (1954) Acute Renal Failure. Blackwood, Oxford*).

During the recovery phase, when the anuria or severe oliguria has given place to a diuresis of pale dilute urine it is essential to remember that the patient is still in a state of renal failure. The dietetic principles should not be relaxed until about a litre of urine is being secreted daily.

The water intake should be increased by a volume equal to the volume of urine passed the day before, and 50 to 75 mEq. of sodium should be added to the intake for every litre of urine secreted, for the kidney at this stage has little power of salt conservation and salt deficiency may easily develop. Many deaths have occurred during this critical phase of commencing recovery.

Sulphonamide Anuria.

Anuria developing during sulphonamide treatment may require special action.

Certain members of the sulphonamide series, especially sulphapyridine, sulphathiazole, and sulphadiazine, are excreted as relatively insoluble acetyl derivatives, and in certain circumstances, depending on the reaction and concentration of the urine, crystals of the acetyl salts are deposited in the renal tubules. The whole renal pelvis and ureter may become blocked by a mass of crystals, blood and debris. This may occur at any stage but always indicates failure to observe proper precautions during sulphonamide treatment. If sufficient fluid and alkali are given, sulphonamide anuria cannot occur, but it must be remembered that patients on sulphonamide treatment are often dehydrated by sweating, pyrexia and vomiting, and therefore an abnormally large quantity of fluid may be necessary in order to ensure an adequate urinary flow. Moreover, sickly patients will not always willingly take enough fluid, and the patient's attendants may not be sufficiently diligent in seeing that fluid ordered is actually consumed.

As a rule the first symptom is renal and ureteric pain, which may be severe. A small quantity of dark chocolate-coloured urine is usually voided, after which, in the worst cases, the flow of urine ceases altogether.

Treatment.—Action must be immediate. Fluids by mouth in large quantities must be given along with alkali as in the treatment of urinary infections described below. If urinary secretion is not re-established within 12 hours, or severe oliguria persists after 24 hours, cystoscopy should be performed, ureteric catheters passed, and the renal pelvis washed out with sodium bicarbonate solution (2.5 per cent.). The sulphonamide should of course be stopped at once, but if the infection for which it was being given persists, appropriate antibiotics can be used. If anuria persists in spite of treatment the condition has probably led to renal tubular necrosis and should be treated as such (*see preceding section*).

Poisoning by mercurial salts.

This is another potential cause of anuria. Unless the specific treatment described below is immediately available several raw eggs in milk should be given at once to precipitate any mercury in the stomach, which should then be thoroughly washed out.

The most effective treatment is by BAL (British Anti-Lewisite, Boots) (Dimercaprol), a substance known chemically as 2:3-Dimercaptopropanol which was elaborated to protect against arsenical poisoning by lewisite. Heavy metals are toxic to biological systems because of their reaction with SH groups of the protein moiety of cellular enzymes to form mercaptides; mercury shares this action. BAL is capable of reactivating enzyme systems poisoned by mercury.

Longcope and Luetscher (*J. Clin. Invest.* 1946. 25, 557) have recorded the results of treatment in 23 cases of poisoning by mercury bichloride. All recovered except one, an early case in the series in which inadequate treatment was given. Nine of the patients had swallowed from 1.5 to 20 G., amounts which are likely to cause death in a high proportion of cases. The authors recommend the following treatment:—

(1) The stomach is washed out with 5 or 10 per cent. sodium formaldehyde sulfoxylate (British Drug Houses) and 300 mg. of BAL (10 per cent. solution in benzyl benzoate and peanut oil) are injected intramuscularly.

(2) One or two hours later, 150 mg. BAL are given, followed by 150 mg. in four to six hours.

(3) Another dose may be given within 12 hours in severe cases, and thereafter two doses per day for the next two or three days, depending upon the patient's condition.

(4) Dehydration and shock are treated by intravenous infusion of saline and dextrose, and by blood transfusion as necessary.

ACUTE NEPHRITIS

In a sense, acute nephritis, even when uncomplicated, is always a medical emergency for it is the opinion of all who study this disease that it is in the first few weeks, and probably only then, that prompt and efficient treatment may affect the outcome. Far too frequently, patients who show the initial signs of acute nephritis, namely oedema, hypertension and hæmaturia, are sent on long bus journeys to wait in the draughty hall of an Out-patient Department. Any patient, at any age, who shows these signs, should be promptly put to bed and kept warm, and if domiciliary treatment is not possible or desirable he should be transferred to hospital by ambulance.

For the detailed treatment of acute nephritis, reference can be made to standard text books; but it may briefly be emphasised that rest in bed should be continued until evidence of acute renal disturbance is over, the oedema and hæmaturia have disappeared, and the blood pressure has returned to normal. Diet should be low in protein, salt and fluid. Milk contains too much of all three and should not be prescribed. About a pint (600 ml.) of sweetened fruit juice and a few biscuits is usually adequate for the first few days.

There are several ways in which the complications of acute nephritis may become medical emergencies. Sometimes these complications are the first signs of disease; more frequently they arise during the early days of a case already diagnosed.

Heart failure in acute nephritis.

The symptoms of acute left ventricular failure not uncommonly occur, resulting either from the rapid development of hypertension or perhaps from the hæmo-dilution which is known to be present in the early stages of the disease.

DIAGNOSIS.—To the uninitiated, and sometimes to the experienced, this may present great difficulties in diagnosis. The

patient is generally a child or a young adult who develops acute dyspnoea and orthopnoea which may be paroxysmal and nocturnal. Cyanosis may be present and congestive signs can be detected in the lungs. Occasionally there may be acute pulmonary oedema if the condition is not relieved. The right side of the heart may become involved as shown by distension of the neck veins and enlargement of the liver. The diagnosis rests on (1) the absence of the ordinary causes of heart failure in young persons; (2) the presence of hypertension which, in a young person, immediately suggests acute nephritis; and (3) red cells, leucocytes and casts in the urine. Many of these patients have only slight oedema, but if it is present in the face and hands, a renal, rather than a cardiac, origin will of course be suggested. It should be remembered that albuminuria (without blood or cellular casts) may occur in any case of heart failure.

Treatment.—This is as in any other case of acute left ventricular failure, with the exception that the mercurial diuretics should not be used. Intravenous aminophylline (see page 598) 0.25 to 0.5 G. (=10 to 20 ml.), is often effective. Morphine can be used and venesection is probably the most valuable remedy of all (at least a pint of blood being taken from an adult patient). Diet (as outlined above) and rest are, of course, essential. In most cases, improvement takes place in a few days and the prognosis is then that of an ordinary case of acute nephritis. Occasionally heart failure is the complication determining a fatal outcome.

Convulsions.

The convulsions which occur in acute nephritis (and also in eclampsia) are not due to any known chemical poison or retained product. There is still some controversy over their exact mechanism but they seem to be related to the acute development of hypertension and hæmo-dilution. Oedema of the brain and arterial spasm are possible factors in their production. The blood urea is often normal, though in severe cases there may be varying degrees of nitrogen retention which bear no constant relationship to the liability to convulsions.

Should convulsions be the first sign of the onset of acute nephritis, the question of diagnosis again arises, especially as a specimen of urine may be temporarily unobtainable. There is nearly always some oedema of the hands and ankles, and hyper-

tension will be present. The retina will rarely show any characteristic features, but spasm of the arteries, hæmorrhages and papillœdema, occasionally occur. A specimen of urine should be obtained (by catheter if necessary) as soon as possible and will show albumin, blood and casts. The presence of a trace of albumin only (i.e., without blood) after a convulsion, would be more suggestive of epilepsy than of acute nephritis. Although convulsions in acute nephritis add to the immediate seriousness of the case and may be a cause of death, they do not add in any way to the gravity of the eventual prognosis; in other words, if the convulsions can be controlled there is no reason why the case should not progress satisfactorily to cure.

Treatment — There are several efficient methods of controlling the fits. Sedatives are valuable, and as they are usually nearer to hand than is the apparatus for venesection and lumbar puncture, it is usual to commence treatment by giving sodium phenobarbitone 0.2 G. (gr. 3) in an adult, intramuscularly. Alternatively, paraldehyde may be given per rectum or by slow intravenous injection (see page 597).

Venesection is often successful. Lumbar puncture should be done with care, using a manometer. If the pressure is high, a few ml. of fluid should be removed, and this may be repeated some hours later. Where there is gross œdema of the brain, the sudden removal of a large quantity of fluid is probably as dangerous as it is in cerebral tumour.

If these measures fail, hypertonic solutions intravenously may succeed. They are better avoided, however, because convulsions probably result from temporary inefficacy of the kidneys to excrete water and electrolytes and there may be a danger in the long run of doing more harm than good. 200 ml. of 50 per cent. sucrose given very slowly would seem to be the least objectionable.

Oliguria and anuria.

In some cases of acute nephritis the urinary secretion is diminished to a few ounces only, or even completely suppressed. This is liable to give rise to panic on the part of the medical attendant and it is common for a series of misdirected attempts at therapy to be applied, in the hope of inducing the kidney to recommence secretion. The administration of large quantities of fluid in acute nephritis is rarely followed by a corresponding

increase in the amount of urine, and may have the undesirable effect of increasing the œdema and seriously disturbing the electrolyte content of the body fluids which the anuric kidney is no longer able to control. The proper course is to keep calm and reiterate the instructions that only a pint of fluid should be given in 24 hours. In nearly every case the kidney will recover spontaneously, and the secretion of urine will be quickly re-established but if diuresis does not occur within 48 hours, the régime described already for the treatment of other types of anuria should be instituted. Decapsulation of the kidney, sometimes advised in the past, is not recommended.

We turn now to three conditions—uræmia, amblyopia and œdema which, although they may occur in acute nephritis, are more commonly the result of long-standing renal disease. All may constitute emergencies under certain circumstances.

Uræmia.

Uræmia is the state which results from failure of the kidney to maintain the normal composition of the body fluids. It occasionally occurs in acute nephritis, and in such cases may be transient. Far more frequently it arises as the end-result of chronic renal disease of all kinds, including chronic nephritis, malignant hypertension, tuberculosis of the kidney, chronic pyelonephritis, polycystic kidney, amyloid disease, hydro-nephrosis, enlarged prostate, and, in fact, any condition leading to gradual destruction of the kidneys or progressive interference with their function. Acute renal failure has already been described.

The most common presenting symptoms of chronic renal failure are anorexia and vomiting. These are accompanied by anæmia, loss of weight, malaise and weakness. The tongue is furred, and the skin dry. A history of polyuria can usually be elicited. Strange as it may seem, all these symptoms may be overlooked or passed off as "biliousness," especially in cases where hypertension is not extreme. The first occasion on which the doctor is called may be when the terminal symptoms of renal failure, namely, drowsiness leading to coma, muscular twitching, convulsions, severe dyspnoea, or acute mania have suddenly developed. It is in this sense that uræmia becomes a medical emergency.

The absolute diagnosis of uræmia cannot be made until blood examination has revealed gross nitrogen retention, i.e., a blood urea

of more than 100 mg. per 100 ml. (normal range of blood urea and non-protein nitrogen 20 to 40 mg. per 100 ml.).

Treatment.—The treatment of renal failure depends on the cause. If there is urinary obstruction as in prostatic disease, this must be relieved in consultation with the surgeon. If the cause is acute nephritis, the treatment is that of the underlying disease. In the far more common cases due to advanced bilateral disease of the kidneys, treatment is purely symptomatic. Methods of purifying the blood by dialysis, although interesting experimentally, have not yet proved sufficiently practical or lasting in their results to warrant general adoption.

Sedatives, therefore, although they may theoretically have an adverse effect on renal function, should not be withheld in these hopeless cases. Phenobarbitone may control convulsions and excitement. Vomiting may be difficult or impossible to control but sometimes responds to dextro-amphetamine in 5 or 10 mg. doses or to chlorpromazine (Largactil) 25 to 50 mg. thrice daily. This may be combined with aspirin and phenacetin if hypertensive headache coexists. True renal dyspnoea, which is very distressing, is caused by acidosis and may often be relieved by giving sodium bicarbonate in doses of 4 G. (gr. 60) every two hours or by intravenous hypertonic sodium lactate solution, e.g. 200 ml. of molar lactate. For uræmic coma there is no effective treatment. Most patients with uræmia will not wish for more than a very light diet. If the underlying disease is very chronic and has hitherto received no adequate treatment, improvement may occur on a diet restricted to biscuits, fruit, and vegetables, with ample fluid (3 litres, or about 5 pints, per day). Milk is not suitable. After a few days the protein can be increased to 40 G. per day. There is no reason to restrict salt in the absence of œdema; in fact, salt reserves may have been much depleted by polyuria and vomiting and occasionally a "low salt syndrome" similar to Addison's disease, with weakness, dehydration and peripheral vascular failure develops. This is shown by a low serum sodium (normal about 144 mille-equivalents per litre, or 330 mg. per 100 ml.) and is amenable to treatment by intravenous hypertonic ($2\frac{1}{2}$ or 5 per cent.) saline.

Another electrolyte disturbance which occasionally appears in renal failure is potassium retention. This is more likely if oliguria is present and if potassium citrate has been prescribed (quite use-

lessly, of course) as a diuretic. The normal serum-potassium is about 3·8 to 5·0 mille-equivalents per litre or 15 to 20 mg. per 100 ml. Values above 7·0 mille-equivalents per litre (28 mg. per 100 ml.) are a danger signal and the patient may die suddenly from cardiac arrest. For treatment *see page 467*.

(*Uræmia resulting from salt depletion is dealt with on page 274*).

Amblyopia and blindness.

There are four mechanisms of visual disturbance in renal disease, hypertension being the cause of each:—

- (1) Hypertensive retinopathy by causing oedema of the papilla, retinal hæmorrhage and exudate.
- (2) Thrombosis of retinal vessels.
- (3) Detachment of the retina.
- (4) Hypertensive encephalopathy. Here a circulatory disturbance, presumably of the visual pathways may lead to temporary blindness without retinal changes.

If the cause of the hypertension is transient or curable, as in pregnancy toxæmia, acute nephritis, and certain cases of unilateral renal disease, the retinal lesions may heal, but if they are far advanced, permanent visual defects will remain.

In hypertensive retinopathy due to advanced renal disease the degree of renal failure usually precludes active treatment by surgery or by methonium compounds, but in some cases, especially of chronic pyelonephritis, hypertension may reach the malignant phase before renal failure has occurred and these should be treated energetically with hypotensive drugs.

Oedema.

Oedema rarely gives rise to an emergency call except when it occurs in the glottis, or the lungs, or when hydrothorax and ascites are sufficiently extensive to cause respiratory embarrassment.

Oedema of the lungs is almost invariably secondary to left ventricular failure, the treatment of which has already been described. Fluid in the pleural and peritoneal spaces can be simply removed by paracentesis.

In gross widespread oedema Southey's tubes will often rid the patient of enormous quantities of fluid. The danger of sepsis is

minimised by antibiotics and the tubes can be left *in situ* for 3 or 4 days. Two tubes are inserted subcutaneously in each leg and allowed to drain through fine rubber tubing. The head of the bed is raised to assist drainage and the whole procedure is "covered" by intramuscular injection of 300,000 units of procaine benzylpenicillin twice daily.

Oedema of the glottis is a very rare, but very dangerous complication of any form of nephritis accompanied by oedema. There is usually oedema of the palate and fauces at the same time, and there may be pain in the throat, dysphagia and aphonia before the acute symptoms of cyanosis and suffocation appear. The treatment is immediate tracheotomy (*see page 569*).

URINARY INFECTIONS

It is only rarely that acute pyelonephritis can be called an emergency, but occasional cases with severe general symptoms require urgent relief. In this type, rigors, pyrexia (often 105°F.) and intense headache are usually the prominent features, the local symptoms such as pain in the back, frequency and dysuria being much less in evidence. The differential diagnosis from meningitis may even arise. On the other hand, dysuria itself may be so distressing as to require immediate treatment.

DIAGNOSIS.—In either case, the provisional diagnosis can usually be made in a few minutes by placing a drop of the cloudy urine on a glass slide, covering it with a coverglass, and examining it microscopically with a dry lens. Pus cells and bacteria in large numbers are immediately apparent. The diagnosis should, of course, be confirmed later by the bacteriological examination of a specimen collected aseptically and the infecting organisms should be tested for sensitivity to antibiotics.

Treatment.—Despite the efficacy of the newer methods of treatment of urinary infections, namely, the sulphonamides and antibiotics, the quickest control of *symptoms* is still to be obtained by the administration of large doses of alkali.

At least 4 G. (60 gr.) of sodium bicarbonate and 4 G. of potassium or sodium citrate should be given every two hours until the temperature is subsiding and the urine is strongly alkaline to litmus. The dosage may then be reduced. [*Potassium citrate should not be given in the presence of renal failure (see page 270).*] There is no special merit in the mixture except that

the citrate gives rise to much less gas in the stomach than does the bicarbonate. If vomiting is troublesome, the medicine may have to be given in quarter doses, at half-hourly intervals. At the same time, providing that vomiting permits, large quantities of fluids should be given, *i.e.* at least 4 litres per day.

Alkalis do not interfere with the antibacterial action of the sulphonamides, and sulphadimidine (Sulphamezathine) 2 G. followed by 1 G. four-hourly may be given four-hourly at the same time. Within a few days, bacteriological reports will be at hand to determine the further treatment. This may include antibiotics such as streptomycin or tetracycline according to the sensitivity tests (*see page 608*). It will be remembered that acute pyelonephritis may complicate renal calculus and other disorders of the urinary tract, and not infrequently occurs during pregnancy. Pregnancy is not a contraindication to the treatment outlined above.

RENAL COLIC

The only difficulty about renal colic as an emergency is to be reasonably sure of the diagnosis. If the pain is severe morphine may be required. This is of course a dangerous drug to give if the case should after all turn out to be one of appendicitis, perforation, or intestinal obstruction, for morphine may obscure the signs and symptoms, and dangerously delay the necessary intervention of the surgeon.

We cannot here discuss the diagnosis in detail. The important points to remember are the characteristics of renal colic; its sudden onset and spasmodic nature; the fact that the patient doubles up or rolls about in the spasms; and the absence of rigidity or muscle guarding. The distribution of the pain may not be characteristic, but in typical cases it strikes from the loin to the groin and often down to the testicle and inner aspect of the thigh. In severe colic sweating and vomiting are usual. A history of previous similar attacks may be helpful, as will a negative history of gastro-intestinal disorder.

The differentiation from biliary colic may be difficult also, but it is of less immediate importance since a powerful analgesic is indicated in both conditions. Mild attacks of renal colic are common in the tropics where excessive sweating leads to dehydration and urinary concentration.

Treatment.—The treatment of the less severe cases is by atropine, rest, and the application of heat to the loin. In severe cases, morphine 15 mg. (gr. $\frac{1}{4}$) or pethidine 50 to 100 mg. is required. When the attack has subsided, an accurate diagnosis as to causation must be made, and will probably necessitate admission to hospital for radiological and cystoscopic examinations.

HÆMATURIA

Hæmaturia, although alarming to the patient, is never a medical emergency in the sense of being in itself dangerous to life. Treatment therefore consists in keeping oneself and the patient calm until such time as the cause can be fully investigated. The value of cystoscopy at the time of hæmaturia should not be overlooked as it may show blood coming from only one of the ureters.

So many of the causes of hæmaturia are serious ones (*e.g.*, carcinoma of the bladder and renal tuberculosis) that to omit thorough investigation or to postpone it until some further symptoms develop, constitutes serious negligence. This applies even though the hæmaturia ceases spontaneously (as it usually does) in a few days.

Occasionally the passage of clots down the ureter gives rise to colic and even to obstruction which will require appropriate treatment including surgical collaboration.

EXTRA-RENAL URÆMIA, THE LOW-SALT SYNDROME AND ALKALOSIS

Apart from the acute renal failure of shock and hæmorrhage a type of uræmia which may be fatal if untreated occurs in states of salt deficiency. It should be suspected in cases where, by vomiting (as in pyloric stenosis) or diarrhœa, body fluids have been lost. It may happen in diabetic coma and also in heart failure treated too enthusiastically by a low salt diet and mercurial diuretics.

Weakness, headache, nausea, vomiting and drowsiness are its chief symptoms. The patient will show the dry mouth, loose inelastic skin and collapsed veins associated with dehydration. The blood pressure will be low. The blood urea is usually 100 mg. per 100 ml. or more and the serum sodium low.

Treatment.—The cause should be sought and remedied if possible but if operation is required for this (*e.g.*, in pyloric sten-

osis) it must wait until sodium deficiency is corrected and renal function restored. In cases due to loss of body fluids it is safe to give 3 or 4 litres of physiological saline by intravenous drip. The first litre can be given quite quickly. Further treatment should be controlled if possible, by estimations of serum sodium, bicarbonate and chloride. If acidosis is present (as it may be in diabetic coma and where intestinal secretions have been lost) some of the saline should be given as $\frac{1}{6}$ molar sodium lactate solution. In cardiac cases where excess of fluid may be dangerous 5 per cent. sodium chloride in correspondingly smaller volume should be substituted. In gastric disorders where the patient has been taking large quantities of alkali, especially sodium bicarbonate, and milk, for a long time, a similar state of uræmia may insidiously develop and should be suspected if such a patient complains of anorexia, nausea, vomiting, lassitude or drowsiness. The blood urea is high. Treatment is by stopping the alkali ingestion and replacing it if necessary by magnesium trisilicate or aluminium hydroxide.

RENAL TUBULAR SYNDROMES

The rarer anomalies of renal tubular function such as the Fanconi syndrome and cystinuria do not constitute emergencies. It has recently been realised, however, that renal disease, especially in the form of chronic pyelonephritis, may give rise to specific tubular damage leading to lack of acidifying power (renal tubular acidosis) or inefficient sodium conservation (salt-losing kidney).

The chronic acidosis due to inability to produce a highly acid urine leads to loss of base, which in turn may give rise to potassium deficiency, or to calcium deficiency and osteomalacia. Potassium deficiency usually manifests itself by attacks of generalised muscular weakness similar to those of familial periodic paralysis and associated with a low serum potassium. Usually they are diagnosed as hysteria and it is important to bear in mind the possibility of potassium deficiency in such cases. They can be cured for the time being by giving oral potassium chloride (3 G. daily) and the syndrome of renal tubular acidosis can then be treated by the continued administration of alkaline salts, *e.g.* 50 to 100 ml. daily of a mixture containing citric acid 140 G., sodium citrate 98 G. in one litre of water: .

The salt-losing kidney manifests itself by a syndrome resembling Addison's disease, with dehydration, hypotension, weakness, low

serum sodium and high blood urea. It does not, however, respond to treatment by cortisone or other adrenal steroids, but only to the administration of salt, which may be given initially as intravenous physiological (or hypertonic 2½ per cent.) saline, and for maintenance as sodium chloride in capsules or with food, about 10 G. daily in addition to the normal diet.

Whereas it is important to realise that such syndromes exist, their proper investigation and initial treatment is generally a matter for well-equipped hospital centres.

ROBERT PLATT.

CHAPTER XVI

Medical Emergencies in Infancy and Childhood

IN this chapter the general and nutritional aspects of medical emergencies in infancy and childhood will be considered first and will be followed by comments on urgent disorders in the newly-born. Special emergencies will then be described system by system.

ACUTE NUTRITIONAL FAILURE

Perhaps the most important group of medical emergencies in childhood is that which includes all types of rapid nutritional failure, with which are frequently associated various dangerous forms of metabolic disturbance affecting mainly the equilibrium of fluid and electrolytes. Many of the patients are infants; the younger the child, the more rapidly does the disorder progress and the more serious is the prognosis.

Several clinical varieties occur—

- (1) Dehydration as in gastro-enteritis, pyloric and intestinal obstruction, and all other diseases and disorders causing vomiting or diarrhoea, *e.g.*, cyclical vomiting.
- (2) Acidosis, ketosis and alkalosis.
- (3) Wasting of body tissues, as in starvation, coeliac disease, fibrocystic disease of the pancreas, septicæmia and other acute infections and advanced tuberculosis.
- (4) "Surgical shock" resulting from trauma, hæmorrhage, burns and scalds, intussusception, volvulus and other types of intestinal obstruction.

In many cases these manifestations overlap; no matter how clearly defined and limited a metabolic disorder may be at its beginning, secondary effects are liable to develop rapidly. Some of these may be compensatory readjustments and care must be taken that the treatment ordered should not oppose any spontaneous physiological reactions that may be helpful to the patient.

Speed in treatment is essential if a critical situation is to be avoided. This is especially true for infants when, for instance, dehydration caused by congenital pyloric stenosis may be combated much more easily and successfully in the first week of illness than, in, say, the third or fourth week.

Treatment will be described under the separate headings given above; but it will be realised that in most cases there is a need for several types of treatment simultaneously. Thus, the vomiting of congenital pyloric stenosis causes dehydration, wasting and, frequently, alkalosis; a child with advanced coeliac disease often shows wasting, dehydration and various avitaminoses; a burnt patient may develop dehydration, acidosis, hypoproteinæmia and "surgical shock"—the latter being due partly to toxæmia with fall of blood pressure, partly to a decrease of blood-protein, and partly to adreno-cortical disturbance.

DEHYDRATION

Severe dehydration is easy to recognise, but the most dependable early sign is progressive and rapid fall in weight. When a baby has lost 5 per cent. of his body weight as a result of dehydration, the physical signs (anxious expression, dryness of the mouth and tongue and depression of the anterior fontanelle) are fairly obvious; a loss of 10 per cent. or more indicates a severe degree of dehydration. In some cases the fluid is lost mainly by vomiting or by diarrhoea; in others the loss takes place chiefly by sweating, or in the expired air. Sometimes the clinical appearance of dehydration is due mainly to a redistribution of fluids, with decrease in the subcutaneous tissues and solid viscera and increase within the intestines—as in intestinal obstruction. These facts are mentioned because they have some bearing on the plan of treatment. The child that has vomited, for example, will be more in need of salt-containing fluids than one whose dehydration results mainly from toxæmia, pyrexia and sweating (*see page 463*). An excess of saline by intravenous injection is liable to cause oliguria and oedema.

Route of fluid administration.

Whenever possible, fluids should be administered by mouth. This requires the services of a conscientious and skilful nurse, who understands her duties clearly and has sufficient time at her disposal. Much patience and perseverance is demanded of

doctors and nursing staff in the management of these cases. In recent years the more general use of intravenous injections—often a life-saving procedure—has had the effect of making doctors and nurses less ready than they were to persevere with oral feeding, but this is the natural way of taking fluids as well as solid food, and even when the child's condition necessitates the giving of parenteral fluids every effort should be made to continue administration by mouth also, with progressive increase from day to day. •

At the same time it cannot be urged too strongly that intravenous infusions are essential for infants suffering from vomiting and diarrhoea (*e.g.* gastro-enteritis), especially when there is evidence of dehydration and acidosis; and for children with encephalitis or meningitis whose swallowing reflex is impaired.

Fluid requirements.

The daily fluid requirements of *normal* children may be tabulated as follows:—

During the first 6 months— $2\frac{1}{2}$ fl. oz. (70 ml.) per pound of “expected”* body weight.

From 6 months to 12 months—2 fl. oz. (60 ml.) per lb. of “expected” body weight.

At 3 years—32 to 50 fl. oz. (1,000 to 1,500 ml.).

At 8 years—50 to 65 fl. oz. (1,500 to 1,900 ml.).

At 12 years—70 fl. oz. (2,000 ml.).

These figures indicate that for each unit of body weight the infant under one year requires approximately three times as much fluid as does the child of twelve.

A dehydrated child needs *additional* fluid to replace that which has been lost and this extra volume can be worked out on the basis of the relation which the weight deficit bears to the expected body weight. For infants it will not often exceed 10 fl. oz. in 24 hours. Thus a dehydrated baby of 6 months, whose expected body weight would be about 15 pounds, but whose actual weight is 12 pounds, will need $37\frac{1}{2}$ fl. oz. ($15 \times 2\frac{1}{2}$) plus $\frac{3}{15}$ of this figure ($7\frac{1}{2}$ fl. oz.), *i.e.*, a total of 45 fl. oz. in 24 hours. As much as possible of this fluid should be given by mouth; but when the child vomits or suffers from an illness requiring alimentary rest, it will be necessary to resort to parenteral administration. It should, however, be noted

* The term “expected” body weight means the average weight of healthy children of the same age.

that a gastric drip (the tube being passed via the mouth or nose into the stomach) or rectal drip (often unsuitable for children under the age of 3 years) may be helpful; the former is particularly useful for immature babies.

Type of fluid.

The most suitable fluid for oral or rectal administration to a dehydrated child is 5 per cent. dextrose in either one-fifth strength physiological saline or one-fifth strength Hartmann's solution*; but a high percentage of sugar (*e.g.*, 10 per cent. glucose or dextri-maltose in water, and flavoured with fruit juice) may be given by mouth to a patient who is not suffering from diarrhoea or vomiting. As the child recovers, a gradual change may be made to human milk, if available, or to a half-cream dried milk mixture, or lactic acid milk.

Older children of highly-strung type, suffering from cyclical vomiting, should at first be offered quite small volumes by mouth, *e.g.*, one dessertspoonful hourly for three hours; then one tablespoonful hourly, with steady increase. It should be administered by a sympathetic but firm individual, preferably not a relative because the psychological aspect is important.

Parenteral routes of administration and types of fluid.

In an acute emergency or when there is vomiting fluid is best given intravenously because the subcutaneous and intramuscular routes do not allow a sufficient volume to be given quickly enough. The fluid requirement of an infant per pound of body weight is considerably larger than that of the older child (*see page 279*); but acute cardiac embarrassment may result if fluid is infused too rapidly into the circulation, especially if solutions containing too much salt are used. Five per cent. dextrose in one-fifth strength physiological saline is a suitable fluid for intravenous infusion. The relatively high dilution of saline recommended should be noted, for though depletion of salt may be an important element in the syndrome of dehydration, especially when it is due to pyloric stenosis, there is danger of over-compensating the deficiency. The infantile kidney concentrates salt badly and therefore if saline is administered in

too high a concentration sodium chloride will be retained, oedema will occur and dangerous respiratory and circulatory embarrassment will result. Sometimes, however, when there has been marked loss of salt by vomiting, it is permissible to start with 5 per cent. dextrose in either half strength physiological saline or Hartmann's solution and after about one-fifth of the total fluid requirement for the day has been administered, to continue with 5 per cent. dextrose in one-fifth strength physiological saline or one-fifth strength Hartmann's solution. During the first two hours the infusion may be given at the rate of 20 drops per minute (which, using the average drip bulb, would be equivalent to about 60 ml. per hour), but subsequently the rate of flow should be reduced to 10 drops per minute (*i.e.*, about 30 ml. per hour). It must be remembered that the volume of the drop will vary according to the dropper used, and it is wise, therefore, to graduate the transfusion bottle so that there is a cross check on the amount of fluid entering the circulation. A scale, inked on a piece of adhesive strapping, will serve very well. It is seldom wise to give to an infant under six months of age more than 20 fl. oz. (600 ml.) in 24 hours intravenously; and when bronchitis or any other respiratory disease is present special care must be taken to avoid giving too large a volume of fluid.

When dehydration is very severe half-strength plasma (20 ml. per pound of expected body weight) should be transfused fairly quickly, *e.g.*, in a period of about two to four hours. The diluting fluid may be one-fifth strength physiological saline or Hartmann's solution and subsequently saline may be infused as recommended above. Hepatitis due to the injection of an icterogenic sample of plasma is an unlikely complication as jaundice from this cause has rarely been encountered below the age of 4 years. In severe dehydration potassium leaves the cells in order to compensate the deficiency of electrolyte in the interstitial fluid and the blood plasma. There is thus an excess of potassium in the circulation coupled with a potentially dangerous deficit in the tissues. The first half pint of intravenous fluid will dilute the potassium in the blood plasma and the total deficit in potassium can then be cautiously made up by giving an appropriate potassium solution. One method of doing this is to use Darrow's solution (potassium chloride 2 grammes, sodium chloride 3 grammes, molar sodium lactate 40 ml., water 710 ml.) of

which 40 ml. per pound of body weight is infused intravenously over a period of 12 hours, *provided the urinary output is adequate*. When oral feeds are begun the potassium may be given by mouth as potassium chloride 1 to 2 G. daily in divided doses. Throughout the process of replacement of fluid an intake and output chart should be kept; this will necessitate as careful an estimate as possible of fluid lost in the vomit, stools and urine. Fluid lost by sweating must not be forgotten.

When the immediate danger of dehydration has been overcome, usually after 24 hours of intravenous infusion, human plasma may again be given with advantage. It is added to the infusion fluid in the proportion of 1 part to 3 or 4, until oral feeding is established. When there is obvious anæmia or when the hæmoglobin is less than 10 G. per cent., a transfusion of compatible blood, preferably fresh, may be given, the total volume administered to infants being 10 to 15 ml. per pound of body weight. An infusion of plasma (10 to 15 ml. per pound) is helpful if a patient must be maintained entirely on intravenous fluids for longer than 48 hours.

WASTING OF TISSUES

There is a decrease of blood-protein in most cases of starvation, whether from lack of food or from diseases such as septicæmia, advanced tuberculosis, coeliac disease, severe diarrhœa, excessive albuminuria, many diseases of the liver, and extensive burns. Although parenteral therapy cannot adequately nourish a patient, children may be tided over a crisis by it. Frequently in such cases the child needs preliminary treatment for dehydration; subsequently plasma or serum may be used. The risk of homologous serum hepatitis must be weighed before plasma is given to older children. The incidence of this potentially serious complication is much reduced if "small pool" rather than "large pool" plasma is used. Not more than 15 to 25 ml. of blood plasma per pound of body weight can be administered with safety in 24 hours.

If it is decided to give a mixture of amino-acids and polypeptides (derived from casein or meat proteins by enzymic digestion), an infusion of protein hydrolysate (*e.g.*, Casydrol) may be given at a rate not exceeding 50 ml. per hour. A half strength solution ($2\frac{1}{2}$ per cent.) is to be preferred as being less likely to provoke thrombo-phlebitis. The daily protein requirement for children under six years is 3 grammes per Kg. of body weight

(i.e., three times the requirement of an adult). This is equivalent to about 50 ml. of half strength Casydrol solution per pound. 25 grammes of glucose should be given at the same time—either by mouth or parenterally—and vitamin B₁ in a dosage of 2 mg. daily by mouth.

SURGICAL SHOCK

Oxygen should be given by inhalation. When shock has been caused by hæmorrhage, blood transfusion is an urgent requirement and it may need to be repeated so long as the patient's systolic blood pressure remains low, say, below 75 mm. Hg. in a child of six years. In other cases infusion of plasma is the procedure of choice and dilution with an equal volume of physiological saline will facilitate its flow through the needle. Dextran, a 6 per cent. solution of high molecular weight polysaccharides, which is isotonic with blood plasma, can be used as an alternative. The risk of homologous serum jaundice is thereby eliminated, but plasma is of greater value if the normal dietary intake of protein has not been maintained. After burns and scalds, when there has been considerable loss of protein, it may be necessary to infuse blood plasma over prolonged periods.

ACIDOSIS, KETOSIS AND ALKALOSIS

When acidosis or ketosis develops, there is usually a quickening of respiration and, in more severe cases, dyspnoea may be obvious. On the other hand, alkalosis is generally accompanied by shallow breathing, amounting sometimes to periodic apnoea. It must be remembered, however, that the clinical picture is often confused by symptoms of the primary illness, *e.g.*, toxæmia, pyrexia, diarrhoea and vomiting and so whenever possible the chemical composition of the blood should be determined. Clearly an attempt should be made to deal as speedily as possible with the primary cause, but valuable and often life-saving assistance may be given by effective treatment of dehydration. When acidosis is present Hartmann's solution (10 to 15 ml. per pound) is suitable for rapid infusion (repeated if necessary) and sodium bicarbonate 1 gramme (15 grains) by mouth four-hourly with syrup of orange 3.5 ml. (m 60) and water to 30 ml. (1 fl. oz.) may be given to a child of one year. For the infant suffering from both diarrhoea and vomiting, Hartmann's solution is the most suitable for infusion. On

the other hand, when alkalosis is present, *e.g.*, in congenital pyloric stenosis or high intestinal obstruction, the loss of chloride may best be made good by infusion of half-strength physiological saline solution. Tetany may be a complication (*see page 251*).

THE NEWBORN INFANT

Inadequate respiration, cardio-vascular collapse, or failure to exercise normal functions such as swallowing or defæcation, may clearly indicate that an urgent situation exists and that the life of the newborn baby is in jeopardy.

Feebleness at birth, characterised by limpness, absence of spontaneous movement, impaired respiration, unsatisfactory colour and low temperature, has as its main causes prematurity, intracranial injury, pulmonary atelectasis and hyaline membrane.

Whether **intracranial injury** is likely to have occurred will, in most cases, be indicated by the character of the labour. Breech delivery, contracted pelvis, prematurity, postmaturity, or a prolonged second stage favour intracranial trauma, perhaps with tearing of the tentorium or the falx cerebri. Anoxia and vitamin K deficiency with consequent hypoprothrombinæmia may be contributory causes.

With infra-tentorial bleeding birth shock is profound. Breathing is shallow and cyanosis occurs in attacks. The pulse rate is slow. Neck rigidity is characteristic and blood will be found in the cerebro-spinal fluid. Medullary compression is liable to occur and the prognosis is poor.

With supra-tentorial lesions, on the other hand, the outlook is rather better; shock is not so marked a feature and hyper-irritability may for a time dominate the picture. Reflex activity is increased. There may be twitchings or clonic movements of asymmetrical character, restlessness and a shrill cry. Frequent yawning is sometimes seen and there may be vomiting. The fontanelle may be tense or have a spongy feel, especially when subdural hæmorrhage has occurred, and exploration of the subdural space through the lateral angle of the anterior fontanelle may be needed. Blood is commonly absent from the cerebrospinal fluid when bleeding is supra-tentorial.

In treating a baby suffering from intracranial birth-trauma it is important to avoid unnecessary handling and oral feeding should be withheld. Treatment in an incubator supplied with oxygen is

often helpful and some clinicians favour intragastric oxygen (see page 577).

Atelectasis is due to incomplete expansion of the lungs at birth. In the absence of intracranial injury causing compression of the respiratory centre, it may be due to prematurity, depression of the centre by sedatives and anæsthetics given to the mother during labour, and blocking of the air passages by inhaled blood or liquor amnii. Breathing is shallow and the cry feeble. Cyanosis is marked. Fine râles may be heard and sometimes consolidation is found by percussion, more especially if pneumonia supervenes. Asphyxia (see page 292) may lead to twitchings or convulsions, but other neurological signs are lacking. Some degree of asphyxia is characteristic of both atelectasis and intracranial injury. (For treatment of asphyxia neonatorum see page 293).

Cyanosis is also seen in congenital heart deformity when careful auscultation will generally reveal a murmur; and in diaphragmatic hernia when the abdomen may seem abnormally empty and the affected side of the chest, usually the left, unduly full, the heart being displaced to the right. Cyanosis is also met with in congenital absence of lung and when the trachea is compressed by a goitre or an enlarged thymus. A tension cyst in the lung causes increasing dyspnœa and cyanosis and displaces the apex beat of the heart; prompt relief follows liberation of the contained air by the insertion of a fine needle through the chest wall. When the results of careful clinical examination are inconclusive great help in diagnosis may be obtained from radiography which will also be a guide to possible surgical treatment.

Twitchings or convulsions, usually the result of cerebral injury, are also met with in neonatal infections, evidence of which may be found on examining the umbilicus, the skin or the respiratory system. Pathogenic *B. coli* and other gram-negative organisms from the mother's vagina are often responsible, in which case penicillin will be of no avail and reliance must be placed on a sulphonamide (page 599), streptomycin (page 605), chloramphenicol (page 606) or tetracycline (page 607). Chloramphenicol is best given as the palmitate in a daily dose of 75 mg. per Kg. Tetracycline is given in a daily dose of 20 mg. per Kg. as tetracycline pædiatric drops (25 mg. to 5 drops). Daily doses should be given in equally divided portions six-hourly.

Convulsions due to hypoglycæmia sometimes occur in the infants of diabetic mothers and are treated by the administration of dextrose (*see page 107*); in an emergency 10 ml. of a 2½ per cent. solution can be given subcutaneously to an infant who cannot swallow, pending the setting up of an intravenous drip of a 5 per cent. solution. In less urgent circumstances, the administration of 1 fluid ounce of 5 per cent. dextrose solution by mouth at hourly intervals may suffice. Tetany is an uncommon cause of convulsions in the newly born and is treated by the intramuscular injection of 5 ml. of a 10 per cent. solution of calcium gluconate: scrupulous asepsis is essential.

Vomiting of liquor amnii and of the meconium or blood with which it may have become contaminated is not infrequent, but persistent vomiting or regurgitation of milk or fluid should arouse suspicion of a congenital anomaly. With atresia of the œsophagus there may be a fistulous opening into the trachea or a bronchus. Fluids will be regurgitated and loud rhonchi will be noted in the main respiratory passages. A number of cases have been successfully treated by operation and diagnosis within the first 24 hours, and before pneumonia develops is essential. Stenosis or atresia of the duodenum or small intestine may likewise account for vomiting from birth. The stomach distends, but the lower abdomen is flat and meconium does not usually appear. Radiography confirms the diagnosis and indicates that laparotomy is needed. **Meconium ileus** is a special type of intestinal obstruction and is thought to be due to lack of trypsin in the intestine consequent upon fibro-cystic disease of the pancreas (muco-viscidosis). The meconium has the consistency of chewing gum and must be removed from the small bowel by operation if the child is to survive. The administration daily of 100 to 200 ml. of a 5 per cent. aqueous solution of pancreatin by mouth may be helpful.

Failure to pass meconium occurs if the anus is imperforate or the rectum is atresic. Digital examination will establish the diagnosis and surgical treatment is imperative (*see page 69*). Meconium ileus, congenital megacolon and stenosis of the small intestine are other causes.

Pyloric stenosis rarely manifests itself within the first week of life and does not have the status of an emergency unless failure to make a diagnosis has allowed the child to become gravely ill, when

the immediate treatment is that of dehydration (*see page 278*) and sometimes alkalosis (*see page 283*).

Hæmatemesis and melæna of the newborn are thought to be due in part at least to vitamin K deficiency: the treatment is described under hæmorrhagic disease of the newborn (*see page 300*).

Icterus gravis (hæmolytic disease of the newborn) is dealt with on *page 298*.

ALIMENTARY SYSTEM

The correct diagnosis of a sudden abdominal disorder in childhood—a frequent problem for the medical practitioner to solve—calls for considerable care and it will not be out of place to make some comments upon the technique of examination and the significance of certain symptoms and signs. First and foremost must be stressed the importance of obtaining a detailed history; this takes time but lessens the likelihood of a mistake. It is necessary to know whether the child has been in full health up to the onset of the present illness, and whether indigestible or unusual food has been eaten recently; what has been the sequence of the various symptoms; whether there has been pyrexia, and whether any symptoms, perhaps nothing more than a slight cough, have occurred to suggest a supra-diaphragmatic cause of the illness. Before the doctor begins his examination he should have obtained so detailed a history that he can visualise quite clearly the successive events which have occurred each day. This demands close attention to the mother's story and steady mental concentration. For instance, merely to ask "Has he been sick?" is not enough; one wants to know "When did he first vomit?"; "How many times has he vomited?"; "How was it related to the abdominal pain?"; and "What has been vomited?"

While it is true to say that many examples of abdominal disorder are caused by relatively mild illness such as simple indigestion, each one *may* represent the beginning of a serious, or potentially serious illness such as peritonitis, appendicitis, tuberculosis, intussusception, volvulus, pneumonia, purpura and visceral bleeding. Abdominal injury causing rupture of the spleen or kidney should also be mentioned because a history of trauma is not always known. Therefore, even when the reported facts point

to the likelihood of some such trivial disorder as an acute gastric upset following dietetic indiscretion, the possibility of one of the serious conditions must still receive full consideration. Extra-abdominal illness such as tonsillitis, rheumatic infection and pneumonia, especially when accompanied by pyrexia, is more likely to cause abdominal pain and an occasional vomit in childhood than in adult life. Hence the routine examination of the child must include a view of the tongue, mouth, throat and ear drums; the taking of the temperature; examination of the heart, lungs, nervous system and urine. Naturally most attention will be devoted to the abdomen and examination of this must be carefully and sympathetically made. If the doctor is not known to the child some time should be spent in making friends. Then the routine examination of the abdomen (inspection, palpation, percussion, and sometimes auscultation) is carried out

Severe abdominal pain in a child is commonly of colicky nature and infants react to it by screaming and drawing the knees up on to the abdomen. Vomiting and distension are other urgent features which may point to acute abdominal disease; they demand a quick decision regarding the possible need for surgical intervention. Little help can be expected from the child himself who will have difficulty in describing and locating his pain; but valuable indications may be noted if his behaviour is closely watched. In addition to a careful abdominal and general examination, the rectum should invariably be examined also. A leucocyte count often helps to differentiate inflammatory from other states.

VOMITING AND DIARRHŒA IN INFANCY

This syndrome is one of the commonest disorders of infancy. It may be mild and readily amenable to treatment without resort to intravenous therapy. Not infrequently, however, a degree of dehydration occurs which threatens life, especially when gastro-enteritis is the cause. Rehydration and the correction of electrolyte imbalance are then urgently called for. Such measures cannot be successfully undertaken in the home and arrangements should be made for the baby's admission to a hospital where the necessary experience and equipment are available.

The illness occurs mainly in artificially-fed children, and there is good reason to believe that it would practically disappear if

breast feeding were more widely practised. Obviously prevention is the ideal, but if that is not possible it is important to identify and remove the cause as quickly as possible. Unsuitable feeding and undernutrition, especially when due to the use of a very dilute sweetened condensed milk mixture, lowers the resistance of the alimentary tract to infection. It is not always realised that underfeeding may cause the passage of small green stools, resembling diarrhoea and that there may be vomiting. Rapid gain of weight, vomiting, and the passage of bulky curds with subsequent loss of weight should suggest the likelihood of overfeeding. The fat of cow's milk is more likely to cause indigestion than the other constituents, and may thus be responsible for diarrhoea and vomiting.

To what extent diarrhoea and vomiting may be due to infection, as distinct from unwise feeding, is difficult to determine in any particular instance, but it is obvious that the more serious cases are infective in nature because the illness may spread to other babies, especially in hospitals and nurseries. In such circumstances tragic emphasis may be given to the truism that babies are generally best cared for by their mothers in their own homes. Sometimes the infection is contracted from an adult who has had an attack of diarrhoea, and in many cases it may be possible to isolate Sonne or Flexner bacilli of dysentery or a pathogenic strain of *B. coli*. The infected adult may be suffering from a common cold rather than an alimentary disease; or pyoderma in the mother may result in severe gastro-enteritis (staphylococcal) in the child.

Generally it is assumed that an infection causing diarrhoea and vomiting produces inflammatory changes in the wall of the intestine but this is not necessarily so, for these changes may be conspicuously absent at autopsy. This is a reminder that a diligent search should be made for evidence of infection elsewhere. For instance, diarrhoea and vomiting may be the first indication of the common cold, otitis media, pulmonary disease or pyelitis.

Management of vomiting and diarrhoea.

In a disease in which the condition of the child may undergo marked fluctuations from hour to hour, it is impossible to lay down a régime suitable for all cases, and much resource is needed in the treatment of urgent symptoms as they arise. It must be

emphasised that skilful and devoted nursing is the keystone of treatment and that the patient should be isolated from other children. The following schedule will serve as a basis for the handling of most cases.

1. Milk feeds should be discontinued in favour of two to three-hourly feeds of half-strength Hartmann's solution (sweetened with glucose). The total fluid intake should be 3 fl. oz. per pound in 24 hours, the calculation being made on actual weight at first, and later, as the child begins to improve, on "expected" weight. A volume not exceeding 10 fl. oz. daily should be added to offset dehydration (*see page 278*).

2. When dehydration is serious, fluid should be given intravenously (Bateman or Keith cannula or plastic polythene catheter, *page 585*). Human plasma 100 ml. diluted with an equal volume of half normal Hartmann's solution with 5 per cent. dextrose is infused first and thereafter one-fifth normal Hartmann's solution in the amounts detailed on *page 279*.

3. At the end of 24 to 48 hours of treatment according to the speed with which vomiting and diarrhoea are relieved it may be possible to give separated dried milk, diluted with Hartmann's solution, three-hourly as follows:—

- (a) For 12 hours, one part milk (reconstituted) to four parts of Hartmann's solution.
- (b) For the next 12 hours, two parts milk (reconstituted) to three parts of Hartmann's solution with progressive increase until full-strength half-cream milk is reached.

It will be necessary to increase the feeds more slowly in some cases, and the need to rest the alimentary tract for a sufficient period requires emphasis. As a general rule it may be said that fluids should be restricted to Hartmann's solution (by mouth or parenterally) during the first 36 hours of treatment. If after this period food by mouth is still not tolerated, protein hydrolysate or plasma must be given intravenously to preserve liver function which is gravely prejudiced by protein starvation. Full-cream milk should not be given too soon after recovery, for the intestine tends to be intolerant of fat after an attack of gastro-enteritis.

4. Drugs are of secondary importance in this disease; sulphadiazine is perhaps the most helpful but penicillin has been disappointing except in the treatment of parenteral infections. Chloramphenicol has been used in daily dosage of 75 mg. per

Kg. body weight (*see page 606*). Phthalyl sulphathiazole may be efficacious when the infecting organism is a dysentery bacillus; the dose is 0.125 G. per pound body weight in 24 hours. Kaolin is without effect. Administration of fat-soluble and other vitamins may have to be suspended while acute symptoms are still present.

OTHER URGENT ABDOMINAL CONDITIONS

Intussusception tends to occur in late infancy, and well-made boys are chiefly affected. There are sudden spasms of colicky pain which cause screaming, and are followed by the passage of a stool. Early vomiting is not unusual. The baby is quiet and apparently normal between the attacks but as the condition advances the stools are found to consist mainly of blood-stained mucus. Examination may show an abnormal emptiness in the right iliac fossa (*signe de Dance*), and a tumour consisting of the intussuscepted ileum and cæcum may be felt in the upper part of the abdomen or on the left side. The apex of the mass can sometimes be felt in the rectum. Distension and other symptoms of intestinal obstruction occur late and it is characteristic of the illness that constitutional disturbance is seldom noteworthy in the earlier stages. Speedy diagnosis is essential, so that surgical treatment can be undertaken before the child's condition deteriorates.

These conspicuous features of intussusception are described because it is important to differentiate the condition from acute ileo-colitis (*page 62*) and from Henoch's purpura (*pages 63 and 179*).

Acute appendicitis (*see also page 89*) is no less common in children than in adults. The clinical features are similar to those met with in older subjects but complications such as gangrene of the appendix and generalised peritonitis usually develop more rapidly. Difficulty in diagnosis arises mainly in a young child who is unable to describe the pain he feels, or in a child whose appendix is abnormally situated. In the latter case, confusion is likely to arise with certain other acute abdominal conditions more or less peculiar to childhood. **Lymphadenitis** (*page 59*) in the ileo-cæcal angle may cause pain in the right iliac fossa with slight pyrexia. A tender swelling is palpable but there is no distension or rigidity, and constipation is not a feature. **Pyelitis** may give rise to pain in

the right side of the abdomen; this diagnosis is suggested when pus cells are found in the urine. A right-sided **perinephric abscess** may be suspected when the inflamed appendix is behind the cæcum, but an appendicular abscess is usually at a lower level than the kidney, and seldom points posteriorly. **Basal pleurisy** (*page 65*) on the right side frequently gives rise to pain in the right iliac fossa, but the signs of pneumonia, with which it may be associated, may not appear until later. The respiratory rate is, however, generally raised, rigidity is not marked and no local swelling can be found.

Pneumococcal peritonitis, which may closely resemble appendicitis, is differentiated from the other forms of acute peritonitis by certain special features. It is rarely seen except in childhood and is much commoner in girls than in boys. A majority of cases are primary and are believed to arise from infection ascending via the genital tract; less frequently it is secondary to pneumococcal infection elsewhere, usually in the chest. The onset may be somewhat insidious, with lower abdominal pain. The accompanying diarrhoea serves to distinguish the disease from peritonitis of other types, for in these, constipation is invariably present. There is rigidity of the abdominal wall and vomiting and distension develop. The face may be flushed and herpes is not uncommon. Treatment is by sulphadiazine (*see page 599*) and penicillin (*see page 601 and also page 608*). In patients who have come under observation in a late stage, one or more loculi of pus may be present and may require surgical drainage. Operation should be deferred until time has been allowed for the abscess to become well separated by adhesions from the rest of the peritoneal cavity.

RESPIRATORY SYSTEM

Cyanosis and dyspnoea of abrupt onset are symptoms which bring certain diseases of the respiratory system into the category of medical emergencies. To these may be added toxæmia, the result of pneumonia or other acute infective process in the lungs or pleuræ, whereby the threat to life is further increased.

Asphyxia neonatorum.

Cyanosis with respiratory depression is the main feature of asphyxia livida; its cause is usually to be sought in obstruction of the placental circulation during birth or of the respiratory passages

after delivery. If unrelieved, cyanosis gives place to pallor (asphyxia pallida) as the circulation fails. White asphyxia, *ab initio*, suggests depression of the medullary centres by drugs or anæsthetics, by intra-cranial injury, or by maternal toxæmia. Thus prolonged and difficult labour, premature rupture of the membranes, prolapse of the cord, breech presentation and the various obstetrical manipulations necessitated by these conditions predispose to asphyxia of the newborn infant.

The possible effect upon the child of every measure intended for the relief of the mother should be carefully weighed. In breech deliveries the body of the infant must be kept warm pending birth of the head, otherwise premature inspiration may occur, with aspiration of fluid from the birth canal. If there is cyanosis the child should be inverted and suction by a small catheter used to remove fluid from the hypopharynx and trachea. Gentle rhythmic compression of the chest wall may then be sufficient to establish normal respiration. If this fails the infant should be immersed in a bath of water at 100°F. and artificial respiration continued (*see page 543*). Oxygen is required if recovery is delayed: it can be administered by nasal catheter or by a *closely applied* soft rubber funnel provided with an expiratory hole (*see page 571*). Alternatively, oxygen can be introduced into the stomach (*see page 577*). In carrying out all these procedures it is of paramount importance to be very gentle, for the condition of the infant is analogous to that encountered in "surgical" shock. Warmth is essential and when breathing is established the infant is placed in a crib heated by suitably protected hot water bottles. Careful observation is necessary even after improvement has set in, for relapse is always possible. In all cases where the survival of the infant is in doubt an incubator must be procured. This not only ensures the maintenance of the correct body temperature but it greatly facilitates oxygen administration (*for risks of oxygen therapy in premature infants see page 32*).

Certain congenital anomalies produce clinical signs closely resembling those of asphyxia neonatorum; for instance, congenital cardiac deformity, malformation of the respiratory passages and œsophagus, tumours of the thymus and diaphragmatic hernia. The outlook varies with the severity and nature of the lesion, and the emergency treatment must be on the lines already recommended for asphyxia.

Bronchitis and pneumonia.

Uncomplicated lobar or broncho-pneumonia rarely constitutes an emergency situation unless antibiotic treatment has been unduly delayed. Penicillin given by injection is the antibiotic of choice; a suitable dosage is 40,000 units of crystalline penicillin in 1 ml. given intramuscularly four-hourly until the pyrexia has permanently subsided (*see also page 601*). Alternatively 300,000 units of procaine penicillin can be injected once daily. It must not be forgotten, however, that in early infancy pneumonia is often caused by the bacillus coli and for this reason it is desirable to add streptomycin. The daily dosage is calculated on the basis of 20 mg. per pound of body weight. *Staphylococcus pyogenes* is not infrequently responsible for a severe form of pneumonia in infancy and early childhood. This organism is frequently resistant to most of the antibiotics in current use. If the organism can be cultured its sensitivities should be ascertained without delay in order that the appropriate agent may be administered, but where the situation is one of great urgency treatment with erythromycin should begin at once (*see page 606*).

Oxygen is of the greatest value in relieving dyspnoea and cyanosis and it is best administered in an oxygen box or tent. Certain of the procedures to be described can be undertaken while the child remains in the oxygen tent.

A small infant suffering from a severe attack of pneumonia may be unable to cough up without assistance the considerable amount of secretion which accumulates in the bronchi. Dyspnoea and increasing cyanosis with obvious rattling in the trachea and pharynx denote a situation of urgency. Secretion must be repeatedly aspirated from the pharynx and the child must, for the time being, be nursed prone and in such a position that gravity can assist the flow of mucus from the trachea. No fluid should be given by mouth.

Viscid secretion can completely block a bronchus and cause the collapse of a lobe or of an entire lung. Displacement of the heart and trachea to the affected side will suggest the diagnosis when X-ray confirmation is not easily obtainable. Urgent dyspnoea is again apparent. Treatment consists in frequent percussion of the back of the chest over the affected lung while the child is in the head-down position, until the mucus plug has been expelled (*see page 131*).

Bronchospasm is another serious complication of bronchopneumonia in infancy. Ephedrine hydrochloride 8 mg. (gr. $\frac{1}{8}$) by mouth or intramuscularly may give relief and can be administered four-hourly. In more pressing circumstances, adrenaline can be administered subcutaneously, minim by minim, until the spasm relaxes. To moisten the atmosphere is often helpful in these circumstances: a steam kettle can be used, or better still, a fine spray into cubicle or tent produced with the help of an oxygen cylinder. If the spray consists of Alevaire (*see page 123*) this will often be found helpful in loosening tenacious secretions.

In staphylococcal pneumonia there is a special tendency to the development of a pneumothorax which is likely to be under tension and to displace the heart away from the affected side. A pyopneumothorax usually supervenes. Insertion of a needle, connected by rubber tubing to a water seal, will relieve the tension and consequent dyspnoea. Pus must be aspirated and an antibiotic to which the staphylococcus is sensitive should be injected into the pleural cavity.

Babies suffering from a severe infection rapidly become anæmic and cyanosis may not be as obvious as would otherwise be the case. Dyspnoea, however, is accentuated and if the hæmoglobin figure is below 50 per cent. blood transfusion should be considered.

Whooping cough. (*see page 312*).

Stridor.

Stridulous inspiration with recession of ribs or of the supra-sternal tissues may occur in acute catarrhal laryngitis and laryngo-tracheo-bronchitis, laryngismus stridulus, acute oedema of the larynx and after inhalation of a foreign body. An acute retro-pharyngeal abscess may cause similar symptoms. Increasing laryngeal obstruction is denoted by restlessness, sweating, pallor and a rising pulse rate. Cyanosis occurs later and if the obstruction is not promptly relieved respiratory failure will follow.

Acute laryngo-tracheo-bronchitis causes intense dyspnoea with supra-sternal or rib recession. The child is usually much relieved if nursed in a humidified atmosphere (*see treatment of bronchopneumonia above*). Bronchospasm is often present as well and ephedrine should be given. *For further measures see page 122.*

Laryngismus stridulus is a manifestation of tetany, and facial irritability, carpo-pedal spasm and evidence of rickets may also

be found; pyrexia and catarrhal symptoms are not usually present. Rapid improvement is obtained by giving calcium gluconate, 5 ml. of a 10 per cent. solution, intramuscularly. The treatment of rickets should be started without delay.

Diphtheria. (*see page 309*).

Inhalation of a foreign body may call for urgent measures (inversion of the patient, etc., *see page 121*) if it is impacted in the larynx. The help of a laryngologist may be needed for its removal through a bronchoscope. If he can be shown a specimen of the suspected foreign body this is a great help.

Inhalation of steam from the spout of a kettle has on occasion led to acute oedema of the larynx necessitating tracheotomy. There is often a latent period before obstructive signs develop and the child must be kept under close observation for twelve hours after such an accident.

An **acute retropharyngeal abscess** is caused by pus tracking behind the posterior wall of the pharynx; it forms a boggy swelling which causes dyspnoea of a choking character. The child adopts a characteristic attitude, sitting with neck extended, the chin supported on the hands, and the elbows on the table. The diagnosis is made by palpation through the mouth and treatment consists in incision of the posterior pharyngeal wall, and child being in the supine position with the neck hyperextended over the end of the operating table. Gravity can then assist the cough reflex to prevent aspiration of pus.

(Chronic retropharyngeal abscesses also occur; they are usually of tuberculous origin but do not cause urgent symptoms and should not be incised).

CARDIO-VASCULAR SYSTEM

Cardiac failure in infants.

Congestive cardiac failure is occasionally met with in young infants. It may be due to a congenital lesion such as persistence of a large ductus arteriosus, to paroxysmal tachycardia, or to idiopathic myocarditis. Dyspnoea and cyanosis are important features and at first sight suggest bronchopneumonia. Rapid enlargement of the liver and the appearance of oedema differentiate cardiac failure from this condition. The response to digitalis therapy is often good. Digoxin 0.125 mg. is administered by mouth and is followed by 0.05 mg. given six-hourly, until

the pulse rate is reduced to 120 per minute. Relief of urgent symptoms may only be temporary and an attempt must be made by means of X-ray studies and electrocardiography to reach a full diagnosis on which further treatment depends.

Acute cardiac failure in older children.

Pallor, vomiting, orthopnoea, tachycardia and an anxious expression are danger signals in a child suffering from the toxic myocarditis of acute rheumatism. The first sound at the mitral area is softened and an apical systolic murmur may be audible. Cardiac dilatation is indicated by epigastric pulsation and an increase in cardiac dulness. Pulsation may be seen in the engorged jugular veins and the liver enlarges and is tender. Cough and basal crepitations indicate congestion in the pulmonary circuit. Pericarditis is an accompaniment of the severer forms of rheumatic carditis, and the soft pericardial friction, best heard towards the base of the heart, should be listened for daily. Subsequently pericardial effusion may develop, causing an increase in the cardiac dulness, and aggravation of all the symptoms and signs already described.

Pericarditis may also occur as a complication of pneumonia, osteomyelitis, and the septic states associated with bacteraemia. In these conditions, the effusion is frequently purulent and its presence may be overlooked on account of profound toxæmia—a serious omission because urgent treatment may well be required.

When rheumatic carditis is of such severity as to cause orthopnoea, the supine position normally enforced in milder cases must be abandoned and the child should be propped up on pillows. Glucose, a heaped tablespoonful to a pint of lemonade, will suffice for nourishment when symptoms are grave; but as improvement sets in, small feeds of Benger's food, bread and butter or plain biscuit with honey or syrup, egg custard or milk pudding are allowed four-hourly. If œdema is present it is wise to restrict milk because of its relatively high salt content.

Restlessness is a serious symptom calling for speedy treatment.

Pain and anxiety are best relieved by opium, *e.g.*, Powder of Ipecacuanha and Opium (Dover's powder) 65 mg. (gr. 1), or Solution of Morphine Hydrochloride 0·06 ml. (1 minim) for each year of life, repeated four to six-hourly as required. Kaolin poultice can be applied to the præcordium. Cough is checked by the Linctus of Codeine. Salicylates are of no value except in carditis associ-

ated with rheumatic fever, when they should be given in doses adequate to control fever and the joint manifestations: overdosage can precipitate oedema of the lungs. Digitalis is often disappointing in the heart failure of acute rheumatic conditions but should be given especially when tachycardia is a marked feature.

A rapidly accumulating pericardial effusion constitutes an immediate threat to life, and should be relieved by paracentesis (*see page 530*). If the fluid is turbid, 250,000 units of crystalline penicillin should be injected into the pericardial cavity, forthwith pending the receipt of a bacteriological report, which will give guidance as to further treatment.

(For heart failure in acute nephritis see page 266).

HÆMOPOIETIC SYSTEM

Anæmia in children and infants is liable to be more rapidly progressive than in adults. Lassitude is an early symptom and jaundice may mask the pallor which would otherwise be readily apparent. Vomiting is of grave significance since it may indicate the onset of a hæmolytic crisis. Urobilinuria, biliuria or hæmoglobinuria may occur according to the severity of the intravascular hæmolysis which has taken place. The child's life is in danger if the red cells have rapidly fallen to 2,000,000 per cu.mm. or if the hæmoglobin has dropped to below 30 per cent. Low figures of this nature are likely to be encountered in icterus gravis neonatorum, acute and chronic hæmolytic anæmia, aplastic anæmia and leukæmia. Hæmorrhage, external or internal, may also lead to serious anæmia; it may be caused by trauma, or by hæmophilia, purpura, and hæmorrhagic disease of the newborn. Exact diagnosis is desirable, but in the absence of adequate laboratory facilities there should be no delay in resorting to blood transfusion when this is clearly needed.

In no condition is accurate hæmatological assessment more necessary than in **hæmolytic disease of the newborn**. Previous examination of the parents' blood will warn us of the possibility. Otherwise jaundice, anæmia or oedema soon after birth and a maternal history of stillbirths or of previous infants suffering with neonatal jaundice, hydrops foetalis or congenital anæmia will suggest hæmolytic disease especially if the liver and spleen are found to be enlarged. The disease is now known to be caused by sensitisation of an Rh. negative mother by her Rh. positive foetus, with the result that maternal agglutinins

lead to hæmolysis of the infant's red blood cells. The appropriate investigations are commonly undertaken by the Regional Blood Transfusion Officer who will require blood from the mother and infant at once. The father's blood should also be submitted when possible. Blood from the parents is obtained by venepuncture: 5 ml. or more is taken into a dry sterile test tube and allowed to clot. The infant's blood is obtained preferably from the untied umbilical cord or from a heel prick; 1.5 ml. is taken into a dry sterile test tube containing a small amount of heparin. The Rh. and ABO groups and where possible the genotypes of the red cells are ascertained from the blood samples and the amount of Rh. antibody in the mother's and infant's serum computed. The Coombs' direct test which is carried out on infants' cells is a particularly sensitive test for maternal antibody and can be completed within 15 minutes. The infant's hæmoglobin and serum bilirubin values are estimated and a blood film is examined.

In the first or second pregnancy the diagnosis is by no means easy; but jaundice with increasing pallor, apparent usually within six hours of birth, should certainly suggest that the necessary blood investigations be carried out. To delay transfusion is dangerous, and this mistake may be made if cases of so-called "physiological jaundice" which is manifest after 24 hours are not subjected to critical consideration. A hæmoglobin value of less than 80 per cent. in capillary blood (or 100 per cent. in cord blood) during the two or three days after birth is indicative of grave hæmolysis and more than one transfusion of Rh. negative blood of the appropriate ABO group may be required. Exchange transfusion is carried out when there has been a previous pregnancy ending in hydrops foetalis or kernicterus, when the baby weighs under 5½ lb. (2.5 Kg.) or when the cord hæmoglobin is below 100 per cent. in full term babies. All untreated babies should be watched carefully and should be given an exchange transfusion if the serum bilirubin rises above 3 mg. per 100 ml. during the first few hours after birth.

Congenital syphilis and **neonatal septicæmia** may both produce a clinical picture which is somewhat similar to that of hæmolytic disease of the newborn. In hæmolytic anæmia of other types, acute or chronic, blood transfusion, repeated as necessary, will prolong life until remission or spontaneous recovery occurs. Special care is needed in young children to avoid overloading

the circulation (*see page 48*) by injecting blood too rapidly; 10 ml. per pound of body weight represents a safe limit, given at the rate of approximately 40 ml. per hour. The total volume of blood needed to bring the hæmoglobin value to 100 per cent. can be worked out thus. The blood volume of infants is 40 ml. per pound of body weight. An 8 pound baby will therefore have a blood volume of 320 ml. If it is desired to raise the hæmoglobin of an anæmic infant of this weight from 60 per cent. by 40 per cent. to the normal 100 per cent. then it is theoretically necessary to give $\frac{40}{100} \times 320 = 128$ ml. In practice 80 ml. of this could be given in the first two hours. The remainder would be given much more slowly over a period of six to eight hours in order that the plasma volume could be appropriately adjusted by interchange with extracellular fluid and by the kidneys. Overloading of the circulation is suggested by distension of the veins in the neck; dyspnoea and even a suspicion of cyanosis would indicate that the transfusion should be stopped at once. Many clinicians are content to raise the hæmoglobin figure to 70 per cent. in the first instance and to give a further small transfusion later if required. It must be emphasised that the hæmoglobin percentage cannot be relied upon as an index of hæmoglobin deficiency when fluid has been given intravenously during the previous 24 hours.

Hæmorrhagic disease of the newborn, usually manifested by melæna, but sometimes by hæmatemesis, or by umbilical or urinary hæmorrhage, is attributable to vitamin K deficiency which leads to insufficient production of prothrombin in the liver. It has been detected occasionally during birth by testing the "meconium" by the benzidine test and showing that it contained blood. Vitamin K therapy should be given (*see page 599*) using not more than 2 mg. of menaphthone (Synkavit) intramuscularly. Blood transfusion should be prepared as it is occasionally necessary also. One fl. oz. of 5 per cent. dextrose solution can be given two-hourly by mouth, but feeding should otherwise be suspended for 24 hours. Hæmorrhage from the vagina without bleeding from any other site ("menstruation of the newborn") may be regarded as physiological and does not require treatment.

(For hæmophilia, purpura and agranulocytosis, *see Chapter IX*).

NERVOUS SYSTEM

Convulsions.

Convulsions frequently accompany acute illness in infancy and early childhood. Only in a minority of cases do they indicate intracranial disease and most frequently they are symptomatic of an infection accompanied by mounting pyrexia. Disorders such as teething, so trivial in themselves as to constitute no threat to life may, by provoking repeated convulsions, produce a state of considerable danger in which the child may die from asphyxia, cardiac failure or hyperpyrexia. It is important, therefore, to treat convulsions symptomatically without delay. Chloral hydrate, in dosage ranging from 65 mg. (gr. 1) under one month to 0.32 G. (gr. 5) at one year, is given by mouth if consciousness is sufficiently regained between spasms to allow the child to swallow. Alternatively, sodium phenobarbitone 0.1 G. (gr. 1½ at one year of age) may be injected intramuscularly. When the fits follow one another in rapid succession an infant should be immersed in a mustard bath which is prepared by the addition of two level tablespoonfuls of mustard, made into a paste, to each gallon of water at 100°F. If the convulsions still persist; paraldehyde 2 ml. is given by intramuscular injection (*see page 597*). A lumbar puncture should be performed in the more refractory cases and when there is reason to suspect that meningitis may be present. A simple enema is administered since relief of constipation may terminate the convulsions. It will also prepare the rectum for the absorption of chloral hydrate (in double the oral dose) or of paraldehyde 3.5 ml (m 60) in saline (*see page 597*) for a child of up to 2 years. In long-continued convulsions the rectal temperature may rise to 105°F. or more and a wet pack may be required to reduce it. The introduction of ice-cold water by tube into the stomach has been found useful; 20 ml. may be so given to an infant. The same principles of treatment, as relevant, apply to status epilepticus in older children. In infants over the age of six months the possibility that the convulsions are associated with rickets and are directly attributable to lowering of the ionised calcium in the blood should not be overlooked. In these cases the fits are a manifestation of tetany and will be promptly relieved by the intramuscular injection of 5 ml. of a 10 per cent. solution of calcium gluconate.

Coma.

Occasionally babies and older children who have previously seemed in good health may become comatose as a result of an unobserved convulsion, or when the head has been injured, causing concussion or meningeal hæmorrhage. In such cases the person responsible for the care of the child may fail to report the injury or convulsion through fear of censure. Coma may also be caused by fulminating meningitis or encephalitis, when neck rigidity will be a feature in some cases. Cerebral hæmorrhage may occur, apart from injury, from a congenital aneurysm of the cerebral vessels or from the rupture of a vessel in a hitherto symptomless cerebral tumour. Cerebral thrombosis particularly of the superior longitudinal sinus, may occur in debilitated infants and may cause convulsions with coma and rapid death. Rapidly fatal coma also occurs in suprarenal hæmorrhage which may result from birth trauma or may supervene in the course of acute infections, particularly meningococcal septicæmia. The sudden collapse of a previously healthy infant with rapid development of ecchymoses and deepening coma, strongly suggests the latter condition (Waterhouse-Friderichsen syndrome, *see pages 201, 256 and 313*). The possibility of poisoning, *e.g.*, by a barbiturate, must not be overlooked.

In children who have recently arrived in this country from abroad, it is well to remember that coma may be a sign of malignant tertian malaria. In all such cases quinine should be given intramuscularly or intravenously without delay; a suitable dose for a child of 5 years would be 0.26 G. (gr. 4) of quinine bihydrochloride, repeated if necessary in four hours.

Paralysis.

It sometimes happens that a child, previously in good health, or following a mild febrile disorder, loses the use of his limbs. In infants a painful condition of the bones may lead to apparent paralysis; before the age of six months this may be the result of **sypilitic epiphysitis**, and after this age, of **scurvy**. Palpation or movement of the limbs causes screaming, and with the help of a careful history and by searching for concomitant signs it should not be difficult to establish a diagnosis. Sudden paralysis should suggest **poliomyelitis**. In this condition signs of slight meningeal irritation are present at the onset, and there is flaccid paralysis with no sensory

involvement. Another cause of paralysis at the same age period is **diphtheritic polyneuritis**. Enquiry will often elicit the history of a sore throat a few weeks previously, the significance of which may not have been fully realised. Some anæsthesia of the hands and feet is usually present, but the most typical sign is palatal paresis with nasal speech and regurgitation of fluids through the nose. Signs of toxic myocarditis may appear and the only safe course to adopt is to insist on strict recumbency until the improvement is obvious. Anti-diphtheritic serum 20,000 units may be given intramuscularly, but at this stage it is practically valueless.

Other forms of peripheral neuritis are rarely seen. **Infective polyneuritis** may lead to an ascending paralysis of the Landry type with involvement of the respiratory muscles. In all forms of paralysis mentioned above it is well to know where a mechanical respirator can be obtained if need should arise (*pages 559 and 640*).

It remains to mention **paralytic chorea**. In this rare form of a common disease there may be suspension of all voluntary movements; but respiration is not affected and there is no danger to life. Diagnosis can usually be made by observing slight choreiform movements in certain muscle groups.

ACUTE OTITIS MEDIA

Earache caused by acute otitis media complicating an upper respiratory infection in a child is a frequent cause of parental anxiety and an urgent call for the doctor.

Examination with an electric auriscope reveals an inflamed eardrum—pink in the early stages, and red and bulging later. The choice to be made is whether to use chemotherapy or not. Sometimes the decision to do so is easy, for there are severe general symptoms or even signs of meningitis. Generally speaking an antibiotic should be used in all cases except those seen late, with established discharge and in whom mastoiditis is probable. Either penicillin (*page 601*), chloramphenicol (*page 606*), or tetracycline (*page 606*) should be given. Within 24 hours the pain is relieved but treatment should be continued until the temperature has been normal for 48 hours though not for longer than six days in all.

The danger of chemotherapy is that it may mask symptoms. Although earache disappears, the drum must be inspected since if it remains opaque and bulging, myringotomy may still be required.

Similarly a purulent discharge and deafness may be the only evidence of mastoiditis, the other signs being masked.

Myringotomy is indicated if the drum remains opaque and bulging. Under general anæsthesia with nitrous oxide or thiopen-

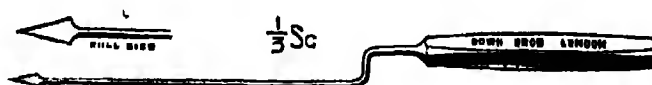


FIG. 28
Myringotome.

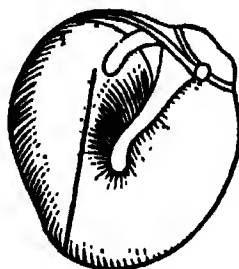


FIG. 29
Showing site of incision
through the right tym-
panic membrane.
(*Pye's Surgical Handicraft.*)

tone, the meatus is cleaned with 70 per cent. spirit, and the largest speculum which gives a full view of the drum inserted. A myringotome (Fig. 28) is then introduced down the speculum and the drum incised from below upwards in its posterior part (Fig. 29). Pus and blood are mopped away. An anæsthetic may be omitted, particularly if the drum is bulging.

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CHAPTER XVII

Emergencies in Infectious Fevers

General considerations.

IN fever practice, problems demanding immediate decision and action arise daily, as every patient admitted is at least potentially infectious. Naturally such problems may occur in any medical practice, private or public, but in fever hospitals almost every patient presents a problem which may be impossible of immediate solution if individual isolation accommodation is limited and the numbers seeking admission are high. Our control of epidemics is still imperfect; an error of judgment or lack of due care may have serious consequences for the individual patient, the immediate contacts or the community at large.

In the field of therapeutics, prompt measures for combating toxæmia as seen in meningococcal invasions or for relieving urgent symptoms in laryngeal diphtheria or bulbar poliomyelitis are required to avert death or prevent permanent disability. Not infrequently, some emergencies prove to be surgical and not strictly medical—a matter of erroneous diagnosis and notification. Acute appendicitis may be mistaken for enteric fever, intussusception for enteritis or dysentery, osteomyelitis or suppurative arthritis for scarlatinal rheumatism, and ruptured ectopic gestation for puerperal peritonitis. It must not be assumed that these errors are necessarily diagnostic blunders on the part of the practitioner. They may be merely abuses of notification used to facilitate admission to hospital.

Emergency measures pending removal to hospital may include the use of specific sera, sulphonamides and antibiotics, but these are best omitted until in hospital lest they hinder the discovery of the causal organism. If they are used the dose, route and time of administration should be stated.

Most medical emergencies in fever practice fall into two main groups: (1) acute toxæmia, (2) urgent dyspnoea. The same patient may show features common to both—as in laryngeal

diphtheria, suffocative pneumonia of measles and whooping cough, and anuric coma of scarlatinal nephritis.

ACUTE TOXAEMIA

Toxaemia may be present from the onset, or develop gradually. Cerebration may appear normal, or all gradations from slight disorientation to manifest toxic psychosis may appear both in the initial stages or as a terminal event. Drowsiness and stupor pass into coma usually accompanied by obvious respiratory arrhythmia and embarrassment. Dehydration and inanition may be early and severe, especially in infants and the aged, or gradual in development with subsequent sudden exacerbation.

DIAGNOSIS.—The best opportunities for successful therapy may have passed before a final diagnosis is possible. Hence to avoid mistakes, the diagnostic net must be large as well as fine.

Investigation of the metabolic disorder consequent upon invasion, as well as the nature of the infecting agent, is carried out more or less on the same lines as in any other clinical condition. The thermometer is useful in excluding non-febrile disorders but may be misleading since normal or subnormal temperature may accompany profound toxæmia in infective disease. Acute septicæmic or meningitic conditions may be closely imitated by uræmia, diabetic coma and cerebral vascular lesions, to mention a few common instances. The presence of a rash, usually of diagnostic help, may mislead, as most eruptive fevers exhibit considerable variation both in the general pattern of the rash and the appearance of individual lesions. This applies especially to prodromal rashes, notably the "bathing pants" hæmorrhagic rash of smallpox and the urticarial and scarlatinal prodromes of measles. Early diagnosis is important also in instituting appropriate administrative measures for the control of spread.

In obscure conditions which cannot be diagnosed outright, it is often found that the optimum time for taking particular specimens or applying specific tests has already passed before the results of the preliminary findings have suggested the true nature of the disease. Foresight is therefore necessary in collecting specimens. Sufficient blood, for example, should be taken (say 10 ml.) to allow of whatever tests are needed at the time, or are likely to be needed later. Half should be allowed to clot (for serum) and the remainder oxalated (for plasma). Throat and

nose washings and cerebro-spinal fluid should be stored on ice or in a refrigerator until delivery to the laboratory. Material, including citrated blood, for examination for viruses is best kept in the frozen state up to -70°C . Any material sent by post must be in a strong box containing absorbent packing and marked "Fragile with care." It must also bear the words "Pathological Specimen" and must be sent by letter and not parcel post.

Treatment.—Treatment of toxæmia may be summed up by saying that it should restore the disordered metabolism to normal as rapidly and completely as possible. In a general way the human body responds to all toxæmias and infections in more or less the same manner. Treatment must, therefore, be largely restorative, supplemented by such prophylactic and therapeutic agents as may be available, commonly immune sera (antitoxic or antibacterial) and/or chemotherapeutic drugs, singly or in combination (*see page 608*). Glucose, hydrolysed proteins and vitamins may be given orally to supplement the ordinary diet, or in special cases intravenously. Dehydration should be dealt with as suggested in the section on *page 463*.

Serotherapy.—As far as is known, antitoxic sera do not influence the action of specific drugs favourably or otherwise, except in so far as they neutralise circulating toxins, and thereby aid the patient to overcome the infection. On the other hand, antibacterial sera may interfere with the action of penicillin (but not sulphonamides) by inducing a dormant ("persister") state in which organism may become temporarily insusceptible to the drug. Clinicians have generally abandoned the use of the antibacterial sera, in the belief that they are now unnecessary and potentially dangerous. This applies also to antitoxic sera with the notable exceptions of diphtheria and tetanus antitoxins.

URGENT DYSPNŒA

When dyspnœa complicates infectious fevers it may be caused by:—

- (1) Mechanical obstruction (*see pages 296 and 310*) from spasm, œdema and exudation (usually infective, but sometimes allergic or traumatic).
- (2) Paralysis of respiratory muscles as in diphtheritic neuritis and poliomyelitis or by spasm as in tetanus.
- (3) Pulmonary disease (*e.g.*, pneumonia).

Paralysis of the respiratory muscles may call for the use of a breathing machine (*see page 559*). Dysphagia is a frequent accompaniment and the operator may be tempted to incline the machine to allow secretions to drain away. This may be done safely for short periods, but prolonged tilting leads to œdema of the brain. Hence it is safer to use the prone position, which calls for minimal tilting or secretions may be withdrawn by a catheter and suction pump. Because of the risk of pneumonia it is wise to use prophylactic chemotherapy. When bulbar failure is present oxygen under pressure may be needed from a mask controlled by a special apparatus such as the Oxford inflator (*page 561*). When there is laryngeal paralysis intubation or tracheotomy may be required and is best done at a special centre (*see page 640*). If secretions continue to accumulate and lead to asphyxia tracheotomy with introduction of a cuffed tube is the best way of controlling them. Adequate ventilation is obtained by a mechanical pump such as a Beaver's apparatus. Feeding may be maintained through an indwelling gastric tube but severe cases require intravenous feeding. Concentrated plasma may correct any tendency to cerebral œdema.

Urgent dyspnoea resulting from bronchitis, laryngo-tracheitis, pneumonia and lobular or lobar collapse may complicate many infectious fevers, particularly measles and whooping cough in the very young. There is usually considerable toxæmia also. Measures to be adopted are described elsewhere, *i.e.*, oxygen therapy (*see page 569*), sulphonamides (*see page 599*), and penicillin by intramuscular injection (Estopen, Glaxo) and by inhalation (*see page 604 and also page 608*).

SPECIAL URGENT FEATURES OF CERTAIN FEVERS

Scarlet fever.

Scarlet fever is now so mild that it is only an emergency when mastoiditis or anuric nephritis suddenly changes the clinical picture. These complications should be very rare even in severe attacks if penicillin is given early in generous dosage (*see page 604*). In toxic or septic attacks intravenous serum may be needed in large doses (*see page 579*), together with penicillin preferably parenterally. A special advantage of penicillin is that it eliminates hæmolytic streptococci from the upper respiratory tract in 4 or 5 days. This allows early release from isolation; the accepted period nowadays is as short as 7 to 10 days—in the absence of complications. For the treatment of nephritis with anuria, *see page 268*.

Sore throat.

When a doctor is called to a patient complaining of a "sore throat," two serious conditions for which treatment must be instituted immediately should be at the back of his mind. They are diphtheria and agranulocytosis (*see page 176*), the former now becoming rare while the reverse holds true for the latter because of the increasing use of drugs toxic to the bone marrow.

Diphtheria.

The nation-wide immunisation campaign pressed vigorously during the war years has yielded a handsome dividend in the virtual disappearance of clinical diphtheria, but while less than 75 per cent. of the population at risk are immunised, severe cases may occur. Antitoxin should be given immediately in silent, obstructive states in which adherent exudate, usually off-white or creamy, but occasionally brownish from presence of altered blood, is present on the tonsils or pharyngeal wall. Loud stridor or a brassy voice suggests non-diphtherial obstruction. Antitoxin should also be given forthwith when adherent exudate is present on both tonsils, especially if it has spread to the palate or uvula, but it is permissible to await the result of bacteriological cultures when it is unilateral, except in young children not immunized. Penicillin or erythromycin may be given in the meantime. Dextran, though useful for shock from burns and wounds, is of doubtful value in toxic diphtheria in which vomiting occurs: plasma is a better nutritive and osmotic agent. Mild tonsillar and nasal cases require 5,000 to 10,000 units of antitoxin intramuscularly, moderate invasions 10,000 to 25,000 units, and late malignant attacks will require 100,000 units or more, at least half of which should be given intravenously (*see page 579*). Whilst adults may need larger amounts, it should rarely be necessary to repeat the dose, especially since penicillin has become available as an adjuvant. Whilst it must be confessed that a clear-cut therapeutic effect is rarely encountered with penicillin, it should always be used in toxic cases. Nourishing food is essential but there does not appear to be any advantage in a high carbohydrate intake with or without insulin, or in intensive vitamin therapy unless for a pre-existing deficiency. Adequate fluids by mouth, by nasal catheter or parenterally if vomiting, dysphagia or coma complicate the picture, using saline or Hartmann's solution, glucose and plasma, are essential in all severe cases. The amounts and concentrations

depend on requirements as revealed by the presence of œdema, and fall in plasma protein level (normal range 6·5 to 8·5 G. per cent.). Any good effect of Injection of Nikethamide B.P., given four to six-hourly in doses of 0·25 to 0·5 ml. for a child of five years, is temporary as is that of adrenaline or ephedrine. Digitalis is inadvisable on account of the frequency of heart block of varying degree in diphtheritic myocarditis. The cold clamminess and pallor of the limbs from peripheral vaso-constriction is best remedied by hot packs, or a hot bath. The effect is usually only temporary. Restlessness, insomnia and distressing præcordial pain may require morphine for complete relief.

Acute circulatory collapse in diphtheria commonly results from heart block (*see page 151*) or from a fall of blood pressure caused by hæmorrhage into the suprarenals (*Waterhouse-Friderichsen syndrome, see page 256*).

Laryngeal diphtheria.

The dreaded *morbus suffocans* of older writers was, until recently, the commonest cause of respiratory obstruction threatening life. The diagnosis from the more frequent coccal laryngitis complicating measles or the common cold can only be made with certainty by direct laryngoscopy. The technique of the examination, suction of false membrane and intubation, can best be learnt in a properly equipped centre such as is available nowadays in most large fever hospitals. A toxic appearance, offensive breath, enlargement of cervical lymph glands and a sanguineous nasal discharge are suggestive accompaniments.

It is still true, and probably always has been, that with equal degrees of obstructive and toxæmic symptoms, the non-diphtherial forms, variously named "croup" or acute laryngo-tracheo-bronchitis, are as serious as those due to diphtheritic infection. In a general way, laryngeal diphtheria responds well to antitoxin and intubation but serum is useless for the other disease. Tracheotomy is generally preferred to intubation on account of the risk of glottic or sub-glottic erosion. (For technique of emergency tracheotomy, *see page 569*). Laryngeal intubation for 12 to 24 hours combined with chemotherapy may obviate the need for operation. Sedation with barbiturates is usually needed to dull the cough reflex. In recent years immunization and chemotherapy have altered the outlook in both conditions, but fatal attacks are still far from infrequent amongst poor, undernourished children.

Sedatives and drugs such as ephedrine may be used to relax laryngeal spasm, but only under *continuous* skilled supervision. If response is equivocal, admission to hospital, especially of babies, is advisable as sudden aggravation of obstructive symptoms may occur, when medical aid may not be at hand. Occasionally the patient is seen for the first time *in extremis*, more often in the casualty department than the practitioner's surgery.

Measles.

The prodromal or pre-eruptive stage of measles may be accompanied by alarming symptoms of laryngeal obstruction ("croup") and by considerable toxæmia; indeed, it is usual for the patient to feel better as the rash appears. While some patients are prone to develop laryngitis with any respiratory infection (even the common cold), the onset of "croup" should suggest a secondary invader. This is usually the hæmolytic streptococcus, but recently coagulase-positive staphylococci (often penicillin-resistant) have been encountered. Only occasionally is the diphtheria bacillus isolated. *Streptococcus viridans*, *micrococcus catarrhalis* and *Hæmophilus influenzae* may acquire pathogenicity and cause changes in the respiratory mucosa already damaged by virus action. Pending culture and sensitivity tests diphtheria anti-toxin and an antibiotic (penicillin or erythromycin) should be given to non-immunes, especially if there has been exposure to diphtheria. The latter will not only help to arrest laryngeal inflammation but may prevent the onset of broncho-pneumonia. Steam kettles or tents and poultices, and even oxygen therapy are of secondary importance. Phenobarbitone in doses of 32 mg. (gr. $\frac{1}{2}$) preferably with sodium bromide 0.32 G. (gr. 5) four-hourly for a child of five years, may avert the need for tracheotomy, but morphine is contra-indicated. Ephedrine 32 mg. (gr. $\frac{1}{2}$) to ease spasm, with or without atropine 0.43 mg. (gr. $\frac{1}{150}$) to dry up secretions may be tried. Caution is needed in using atropine since the resulting tenacious plugs of muco-pus may cause pulmonary collapse. An X-ray examination is indicated in all but the mildest cases. For measles encephalo-myelitis see page 317.

Mumps.

Orchitis, still common in males after puberty, may be obviated by diethyl stilboestrol 1 to 2 mg. four times a day for five days. The fully developed condition has responded to doses of three to

four times this amount, so that incision of the tunica albuginea has become an obsolete operation.

Whooping cough.

Whooping cough has replaced measles as the worst enemy of childhood, mainly by reason of the severe irreparable damage which may be inflicted on the lungs even by a mild attack. The severe bouts of coughing (*see page 145*) may be relieved by Mist. Codein. pro. Inf. N.F. 4 ml. (1 teaspoonful) up to 4-hourly for a child, or dihydrocodeinone (Dicodid) 22 mg. (gr. $\frac{1}{3}$) by mouth for an adult, or phenobarbitone 16 mg. (gr. $\frac{1}{4}$) twice daily in a mixture. Some workers find Seconal (Quinal Barbitone B.P.) suppositories 0.13 G. (gr. 2) particularly good in babies. A warning must be given against the use of atropine or belladonna since this, by causing inspissation of muco-pus, may lead to bronchial obstruction and lobular collapse. These patients should be nursed prone or semi-prone and suitably tilted instead of in the conventional propped-up position. Timely aspiration through a bronchoscope may avert subsequent development of fibrosis and bronchiectasis.

Reports on broad spectrum antibiotics (*see pages 606 and 607*) suggest that they may have a permanent place in treatment, at least in preventing respiratory complications. Erythromycin is effective and does not suppress intestinal commensals.

Bronchopneumonia complicating whooping cough is a medical emergency and calls for prompt antibiotic therapy. Streptococcal infections respond best to penicillin (preferably Estopen) but in mixed infections it may be combined with sulphonamides (*see page 608*) or the newer antibiotics.

In infants especially, convulsions (*pages 285 and 301*) and gastro-enteritis (*page 289*) are urgent complications and constitute specially dangerous threats to life.

Meningococcal infections.

Sudden onset of coma, especially with a purpuric rash, should suggest "spotted fever," even before the onset of meningitis. Blood should be taken for culture, putting 5 ml. into 50 ml. of serum broth and prompt intensive sulphonamide therapy (intravenous if necessary) started. Intra-theal injection of sulphonamides is strictly contra-indicated in any form of meningitis. There is no evidence that penicillin gives better results. With modern treatment the case mortality should be well under 10 per cent., being very low (under 1 per cent.) in young adults, but remains high in

infants and those over 50 years. Nevertheless fatal fulminating attacks with extensive purpura, adrenal hæmorrhages (*see Waterhouse-Friderichsen syndrome, page 256*), and, very occasionally, encephalitis, may occur at any age. Occasionally blocks occur at the foramen magnum, the iter, or over the cortex, necessitating the special measures which are more frequently needed in other pyogenic forms of meningitis.

Pneumococcal, streptococcal, and staphylococcal meningitis.

Prompt intensive antibiotic therapy is required as the prognosis worsens with delay in starting it. Penicillin (2 to 4 mega units daily) should be given systemically (*see page 599*) and intrathecally (25,000 units of crystalline penicillin in 5 ml. of water diluted with 5 ml. of C.S.F.) and combined with sulphonamides by mouth or intravenously (*see page 608*). If blockage occurs, the spinal canal should be washed out with saline through needles inserted at the cisternal and lumbar sites. Introduction of penicillin into the ventricles and the subarachnoid space or over the hemispheres may be required in desperate cases. Not more than 10 ml. should be inserted at a given site, daily, or on alternate days until recovery. The help of a neurosurgeon should be sought and any focus of infection such as otitis should be dealt with. If drug resistance should occur, a broad spectrum antibiotic, preferably chloramphenicol, is advisable.

Influenzal (Pfeiffer) meningitis.

This serious disease, fortunately infrequent in this country, but common and on the increase in America, is an emergency calling for intensive therapy. Streptomycin is effective but early development of drug-fastness is a serious disadvantage, even with full dosage of 25 to 50 mg. in 3 to 5 ml. saline intrathecally every 24 hours and 20 to 40 mg. per lb. body weight intramuscularly in 4 divided doses. Recent experience decisively favours chloramphenicol as the best antibiotic (*see page 606*) as it rapidly reaches the C.S.F. in effective concentration. Treatment should continue until resolution occurs, generally in 5 to 7 days. Longer treatment or even a second course may be needed occasionally. In severe or late cases one or two doses of streptomycin may be given intrathecally in combination with chloramphenicol.

Tuberculous meningitis.

The chief diagnostic difficulty once meningitis has been recognised and lumbar puncture performed is to distinguish between

tuberculous meningitis, post-infectious encephalitis, non-paralytic poliomyelitis and lymphocytic chorio-meningitis. The history and chest X-ray may be helpful but the final decision rests on finding tubercle bacilli in the C.S.F. A glucose level below 50 mg. per 100 ml. and a chloride level below 600 mg. per 100 ml. are strongly suggestive but these findings are seen occasionally in aseptic meningitis. Recent convulsions tend to raise the C.S.F. sugar and isoniazid, a reducing agent, to lower it. In doubtful cases the bromide permeability test may help (*Lancet*, 1954. 1. 700).

The long and difficult nursing makes transfer to special centres advisable but treatment may be started elsewhere as an emergency measure after first ascertaining the routine at the centre. Although many recoveries are claimed from the use of intramuscular streptomycin and oral isoniazid without intrathecal injections the general opinion at present is that combined intrathecal and intramuscular streptomycin therapy gives the most consistently good results. The daily dose should be at least 20 mg. per pound intramuscularly and 50 to 100 mg. according to age intrathecally for a total of at least 60 days. If subarachnoid block develops streptomycin may be given into the ventricles through burr holes, but this is necessary only in special cases. Dihydrostreptomycin is more irritating than the calcium chloride complex and should be avoided intrathecally (*see also page 605*). Isoniazid (iso-nicotinic acid hydrazide) 5 mg. per pound (10 mg. per Kg.) daily has replaced *para*-amino salicylic acid (P.A.S.) in meningitis as a means of obviating drug resistance. It is important not to inject streptomycin intrathecally in the pre-paralytic stage of poliomyelitis with which tuberculous meningitis may be confused. It may be added, to warn those who may be tempted to try cortisone, that it diminishes rather than increases resistance to tuberculosis.

Typhoid fever.

Chloramphenicol has superseded anti-serum as well as penicillin and sulphathiazole in the treatment of typhoid fever and has permitted greater freedom in diet. An average course for a 150 lb. patient is 1 G. orally in 0.25 G. capsules followed by 0.5 G. every four to six hours for five days according to whether diarrhoea or constipation is present. Children can tolerate and probably need proportionately larger doses, especially when diarrhoea is present. As children cannot swallow capsules and the taste is extremely bitter, a suitably flavoured suspension is required. After

an initial fall of temperature which may be accompanied by temporary aggravation of symptoms a moderate rise may ensue for one to three days, but subsequent satisfactory progress while therapy is maintained is the rule. However, as the action is merely bacteriostatic, the relapse rate is high and can be countered by prolonged administration (14 to 21 days) or preferably, by two or three courses each of 5 days with 4 to 5 days' interval between them. Drug resistance does not appear to follow and response in relapse is even more clear-cut than in the primary attack. On the other hand, the drug appears to have little action in influencing the carrier state, whether biliary, intestinal or urinary, either in preventing or curing it. It is a wise precaution to give vitamin B complex and vitamin K (*see page 599*) when the drug is given for periods over a week. Chemotherapy tends to retard the development of immunity and small doses of T.A.B. vaccine (0.1 ml. every 5 to 7 days) may be given to offset this. Sometimes tetracycline (*see page 607*) succeeds where chloramphenicol apparently failed.

Hæmorrhage, which occurs in 5 to 10 per cent. of cases untreated by specific chemotherapy, varies from a mere oozing to the sudden loss of several pints, manifested by pallor, rapid thready pulse, sighing respiration and a fall of temperature. There may be dull pain or a sensation of something giving way in the abdomen. If the patient is constipated, blood may be passed as tarry stools only after a long interval. Treatment is on the usual lines for shock, by morphine and blood transfusion.

Perforation is nowadays a rare complication of typhoid because the improved dietetic regimen has almost eliminated severe meteorism but it may occasionally complicate an apparently satisfactory case. The usual onset is with sudden pain, followed by tenderness and rigidity of the lower abdomen, but these features may be masked in the presence of severe toxæmia. After a brief fall in temperature and apparent improvement following the initial shock of perforation the temperature rises with the onset of generalised peritonitis. Increasing distension, rapid respiration and the Hippocratic facies appear, and unless operative measures are quickly undertaken death usually follows in 36 to 48 hours. Spontaneous cure has been recorded, but this is more likely with specific chemotherapy.

Dysentery-Salmonella infections.

(Symptomatic treatment is described on *page 289*.) When acute diarrhoea is shown to be caused by one of the dysentery-salmonella organisms a soluble sulphonamide (preferably a triple combination, *see page 601*) should be used ; when diarrhoea has ceased phthalyl-sulphathiazole or phthalyl-sulphacetamide (Enterocid) is substituted, the whole course lasting 4 to 6 days. The dose for an adult is 4 G. followed by 2 G. four-hourly. These drugs are non-toxic in these amounts and the effective dose is smaller than that of sulphaguanidine or sulphasuxidine. Chloramphenicol is more active against dysentery and salmonella bacilli than are the sulphonamides, but not all strains are equally susceptible ; non-susceptibility is more common, and exists to a higher degree in Salmonella organisms which may be refractory to every known antibiotic. A combination of streptomycin and tetracycline is worth a trial in persistent carriers.

Smallpox. (*see also pages 365 and 611*)

During the war years and subsequently, smallpox has been introduced on numerous occasions into Great Britain but only once, in 1942, in Scotland, did it establish itself sufficiently to require mass vaccination. The short journey by air from endemic areas and the occasional tendency for oriental smallpox (*variola major*) to break through the protection afforded by vaccination as practised at present, combine to increase greatly the risk of importation. The highly modified form with few lesions may be disregarded by patients and unrecognised by doctors. It can, however, cause malignant attacks in the unvaccinated, the source of which may not be readily traced. Severe influenzal symptoms, amongst which backache is prominent, followed by a few papules usually on the face but which may commence on the trunk and may not pustulate, should be regarded with suspicion and the nearest Public Health Laboratory (*see Appendix I page 622*), consulted with a view to isolating the elementary bodies and performing the complement fixation test. The doctor should communicate his suspicions to the local Medical Officer of Health, who, in the event of doubt, has at his disposal a panel of consultants, and facilities for sending specimens (*see page 630*) to the virus reference laboratory (Colindale 7041); the Bacteriological Department, Liverpool School of Hygiene (Royal 6022); the Brownlee Laboratory, Ruchill Hospital, Glasgow, N.W. (Maryhill 3232)

or the Bacteriological Department, University New Building, Edinburgh, 1 (Edinburgh 42542). Suitable materials are the contents of vesicles, scrapings of the base of papules or even crusts. The result of the complement fixation test is available in 24 hours and is later confirmed by culture on the growing chick embryo. In the treatment of malignant attacks, characterised by prostration and vomiting, intravenous dextrose-saline and plasma, convalescent serum (variola or vaccinia) should be employed, together with sulphathiazole and penicillin or tetracycline to combat the effects of staphylococcal pustulation. There is no evidence that any antibiotic is active against the smallpox virus. Gentian violet and di-bromo-propamidine, both in 1 or 2 per cent. solution or cream, are valuable in controlling skin sepsis, both being bland and stable with the tendency to sensitisation.

ENCEPHALITIS COMPLICATING INFECTIOUS FEVERS

When encephalitis is thought to be complicating an infectious fever a knowledge of the likely time of onset of encephalitis is helpful. The usual figures are: —

MEASLES.	Third to fourteenth day of illness, as temperature falls and rash is beginning to fade, <i>i.e.</i> , about the third post-eruptive day.
RUBELLA.	Three to six days after the appearance of the rash.
VACCINIA.	Five to twenty-three (usually nine to thirteen) days after the vaccination.
SMALLPOX.	One to twenty-eight (usually eight) days after the appearance of the rash.
WHOOPING COUGH	
"ENCEPHALITIS."	Two to seven weeks after onset.
MUMPS.	Within one week of the parotitis.
CHICKEN POX.	Five days after the rash.

Encephalitis, following various virus diseases, including vaccination, was made notifiable on 1st January, 1950.

In general, the severity of this complication is related to the severity of the primary disease.

Measles encephalo-myelitis.

Although improved nutrition and hygiene of the susceptible population and timely chemotherapy have resulted in a striking fall in the complication and fatality rates of measles in recent years, a notable exception is acute disseminated meningo-

encephalo-myelitis which seems to be as common, though perhaps not as fatal, as ever. It may complicate mild attacks, the incidence being about 1 in 2,000 cases and the case mortality about 10 per cent. The onset is marked by headache, vomiting and rapidly increasing drowsiness and stupor 3 to 14 days after measles begins. Exceptionally it may appear before the rash. Cerebral and spinal forms have been described. Diagnosis is confirmed by lumbar puncture which reveals a fluid usually under slightly increased pressure, containing excess protein and mononuclear cells (20 to 100 per cu.mm.). Glucose and chlorides are not reduced, thus differentiating the condition from tuberculous meningitis. Lymphocytic chorio-meningitis and non-paralytic poliomyelitis may be difficult to exclude if these infections are prevalent at the time.

Treatment.—Lumbar puncture and intravenous hypertonic (25 per cent.) dextrose or concentrated (4 or 5 times normal) human plasma may be used if the C.S.F. pressure is high. Corticotrophin has been used with success the rationale being that the condition is an allergic encephalopathy rather than an encephalitis. The patient should be propped up although slight tilting in the prone position is warranted when pocketing of secretion occurs. For an infant, 5 per cent. calcium gluconate should be used as the higher concentration may cause muscle necrosis. If the disease progresses, convalescent measles encephalitis serum, if available, or measles convalescent serum, should be given intravenously in doses of 10 to 50 ml. according to age. Failing this, gamma globulin (*see page 514*) may be tried. The contents of one bottle, 250 mg., should be dissolved in 3 ml. of sterile pyrogen-free water and injected. Older children can have 500 mg.

Encephalitis complicating rubella, smallpox, vaccinia, whooping cough, mumps and chicken pox should be treated on the same general lines. Sometimes, especially in mumps, encephalitis may precede or even occur in the absence of the primary disease. The complement fixation test establishes the diagnosis.

Prognosis in encephalitis prompts urgent parental inquiries. It is variable and especially so in the encephalitis of smallpox. Encephalitis of measles has a 10 per cent. mortality. Vaccinial encephalitis is fatal in about 40 per cent. of cases. Pertussis encephalitis is a doubtful clinical entity, the previous complications being vascular in origin or due to hypoxia. Case fatality is between 30 and 50 per cent. Encephalitis with subsequent lasting encephalo-

pathy has occasionally followed pertussis inoculation. The encephalitis of rubella, chicken pox and mumps is rarely fatal.

EMERGENCY ASPECT OF PROPHYLAXIS

- The need for urgent protection against infection may, in the young and debilitated, constitute an emergency.

Smallpox. (*see also pages 365 and 611*)

Immediate contacts should be vaccinated and may be allowed to travel to their homes but the local Medical Officer of Health should be informed forthwith; the earlier it is done the more likely is it to be effective. Mass vaccination is only called for if the disease is of the major variety and has established itself in the community.

Diphtheria.

The emergency prophylactic measures to be adopted depend on the circumstances. If there is only one case, no action need be taken beyond careful watching of contacts for a week. If further cases arise they should be isolated, and contacts Schick tested. Positive reactors should be actively immunized. If cases continue to occur or if new entrants are introduced to the community, combined active and passive immunization should be conferred as an emergency measure to abort the threatened epidemic. Indiscriminate swabbing of contacts is nowadays discouraged and is unnecessary if the herd immunity is high from antecedent immunization. School closure and exclusion of contacts are measures rarely practised nowadays, being superseded by immunization and close supervision for early signs of the disease. Occasionally Schick negative subjects may develop diphtheria but the disease is then nearly always mild. Penicillin or erythromycin is preferable to antitoxin in such cases, being safer and more likely to effect early elimination of the organism.

Tuberculosis.

The use of B.C.G. vaccine in immediate contacts who are Mantoux negative would now rightly be regarded as an emergency measure.

IMMEDIATE PROTECTION BY PASSIVE IMMUNIZATION

This may be called for as an emergency procedure because the exposed person is already ill or debilitated. Passive immunity is short-lived, providing 10 to 14 days' complete protection and

alleviation of the disease if contracted in the subsequent week or two. It is available for the following diseases:—

DIPHTHERIA.—Give 2,000 to 5,000 units of anti-diphtheritic serum intramuscularly according to age. It may be combined with active immunization by giving 0·5 ml. alum precipitated toxoid followed four weeks later by 1·0 ml. One of the more bland preparations, *e.g.*, T.A.F., should be used if poliomyelitis is prevalent, the injection being subcutaneous and not intramuscular.

SCARLET FEVER.—If protection is desirable in particular circumstances procaine benzylpenicillin (*see page 602*) once a day for two or three days suffices. The use of antitoxin for this purpose has been virtually abandoned.

- MEASLES.—Give convalescent serum 0·2 to 0·3 ml. per lb. body weight in the first five days after exposure. As this may not be readily available, an alternative is to give parents' blood (0·5 to 1·0 ml. per lb. body weight) provided they have had measles. Reduction of the dose or postponement to the latter half of the incubation period usually modifies the attack but as an emergency measure complete protection is desirable. Gamma globulin (issued by the Central Public Health Laboratory, Colindale, London, N.W.9. Tel. Colindale 7041) is to be preferred to unaltered serum because it is more stable and its effective dose is smaller. It is also virtually free from the risk of transmitting the virus of hepatitis. A dose of 250 mg. in 3 ml. (equivalent to 5 fl. oz. of blood) usually protects infants but up to double this amount is needed for older children in whom modification is a more probable and more desirable result.

Convalescent serum is also available against rubella, chicken pox, mumps, whooping cough and poliomyelitis, but only against measles is it uniformly reliable. The protective titre of convalescent pertussis serum may be enhanced by previously inoculating with vaccine donors who have had the disease (hyperimmune serum). Alternatively, the serum of immunized rabbits may be employed, or the gamma globulin normally used in measles prophylaxis, in which event the dosage should be increased by 50 to 100 per cent. as donors are less likely to have had whooping cough than measles previously. The risk of transmitting hepatitis is too serious to warrant indiscriminate use of immune serum, or indeed of human blood or any of its derivatives, except possibly

gamma globulin. These substances should only be used when it would be considered unsafe to withhold them.

PASSIVE IMMUNIZATION OF PREGNANT WOMEN.

An impressive body of evidence, not yet amounting to final proof, has been collected to the effect that certain virus infections (rubella, measles, mumps and influenza) in the mother during the early months of pregnancy may, and in the case of rubella usually does, damage the foetus. Cataract, deafness, congenital heart disease and other abnormalities may result.

In the first three months the risk is so great that if a pregnant woman contracts rubella, therapeutic abortion should be considered. Most large fever hospitals hold stocks of dried convalescent sera against most of these diseases. The average dose is 20 ml. preferably given in the first three days after exposure; if given later, the dose should be proportionately increased. On account of the possible risk of homologous serum jaundice, which cannot be entirely foreseen or avoided, human serum, especially when taken from a large pool, should not be administered indiscriminately or unnecessarily. The use of the gamma globulin fraction would allow reduction of the amount inoculated and diminish the risk of jaundice. The dose to prevent rubella is 750 to 1,000 mg.

ISOLATION AND QUARANTINE

(For Infectious Diseases in General Wards see page 513)

The recognised rules of quarantine have been considerably relaxed in recent years and no useful purpose can be served by trying to formulate rules and regulations to meet all contingencies.

Up-to-date recommendations regarding isolation and quarantine of patients in hospital and their discharge stress that particular circumstances must govern the appropriate action whether it affects the individual or the group. Legally, contacts and carriers cannot be controlled or their liberties restricted apart from prohibition of the handling, preparation and cooking of foodstuffs by carriers of intestinal pathogens.

Generally speaking, the patient is not infectious during the incubation period and no restrictions on movements are commonly applied. (Infectivity has been proved in the last 24 hours' incubation in chicken pox, and in the last 2 or 3 days in enteric fever. It may conceivably occur similarly in other infectious diseases, includ-

ing poliomyelitis). The quarantine period, rarely applied in strict form nowadays, is the maximum incubation period with one or two days added in case the initial phase of the attack is overlooked. Some measure of supervision during the whole incubation period is advisable. Contacts may contract the disease early in the supposed incubation period because they had been exposed to an undetected case or carrier as well as to the first recognised case. Smallpox is the classical example of the official policy of surveillance as opposed to strict imposition of isolation in quarantine, and is almost invariably successful when combined with prompt vaccination. For common infectious diseases such as measles, scarlet fever, whooping cough, and diphtheria, similar prophylactic measures are available supplemented by search for the causative organism when practicable, and isolation of proved carriers. Mumps, rubella and chicken-pox may give rise to considerable trouble, especially in schools, barracks, and similar semi-closed communities, not because of the severity of the disease but because the prolonged incubation periods and relatively low infectivity may lead to persistence for months and interfere seriously with essential activities. Sufficient immune serum is rarely available to protect all immediate contacts. Infantile paralysis (*see also page 213*) may present a serious problem in similar communities, with even more tragic consequences. Again, the appropriate action depends on the circumstances, and what is best for the individual may not necessarily be in the interests of the population at large. Break up of a school is the shortest way to abort an outbreak and may be attended by little risk if susceptibles (generally under 25 years) are temporarily removed from the respective homes to which the contacts are dispersed. On the other hand, if the contacts belong to the poorer classes with large young families this measure might prove highly dangerous in converting a single focus into multiple potential foci. Recent experience in Australia has shown the "stay put" measure to be the most effective method of preventing epidemic spread whether it affects individual families, isolated communities, or large towns. During periods of epidemic prevalence operations on the upper respiratory tract and especially tonsillectomy and tooth extraction should be avoided; if operation is imperative a protective dose of convalescent poliomyelitis serum is advisable. Similarly injections likely to cause tissue destruction, such as A.P.T., should be postponed as subsequent invasion may be facilitated by the *locus minoris resistentiæ* so provided. The

tendency for strenuous exercise such as swimming to favour invasion and dissemination in the body with resulting severe paralyses is well recognised; infection by this means is doubtful, but close aggregations of susceptibles during an epidemic is obviously undesirable.

EMERGENCY ASPECTS OF DISINFECTION

The term disinfection implies destruction of the *materies morbi* and not sterilisation. In recent years current disinfection from day to day, and virtually from minute to minute, has largely replaced so-called terminal disinfection at the end of the illness. If the former has been efficient, the latter becomes largely superfluous.

Current disinfection.

The measures employed naturally depend on the resources available. All contaminated discharges and excreta should preferably be treated for one hour with 2·5 per cent. Cetrimide B.P. or 5 per cent. lysol solution in an amount equal to that of the material to be disinfected. Instruments, crockery and bed-pans should be boiled for 10 minutes, and fabrics treated with current steam at 230°F. for 15 minutes. Some instruments, especially electrical, are more conveniently disinfected by exposure to ultra-violet light at one foot for one minute.

Terminal disinfection.

The end of the illness is a suitable time for disinfecting mattresses, blankets and room furnishings. No special measures are needed for walls and furniture beyond washing with soap and water. Cleansing is facilitated by addition of a detergent. Cetrimide is probably best, being bactericidal as well as detergent and is unaffected by soap. Viruses tend to die rapidly after leaving the body but common organisms such as streptococci, staphylococci and tubercle bacilli may survive for months and sporing organisms such as tetanus for years in dust and soil. A rapid emergency method of disinfecting a room is to use resorcinol, formalin or propylene glycol fog, but dust must first be removed from the atmosphere and from the surfaces of the room and its contents.

Transport of infectious cases.

Ambulances are sprayed with antiseptic solution (carbolic acid, formalin or hypochlorite). Local authorities have arrangements for disinfecting taxis and other public vehicles should they be

contaminated. Bodies of those dying from infectious diseases should be taken direct to the burial place.

Burial. .

It is not considered necessary to disinfect hearses. It is, however, advisable that undertakers who have to handle corpses dead from smallpox should be vaccinated or re-vaccinated. Successful vaccination may be accepted as conferring protection for five years. All unsuccessful vaccinations thereafter should be followed by repeat attempts after each exposure to the disease.

WILLIAM GUNN.

CHAPTER XVIII

Emergencies in Tropical Medicine

General Considerations.

PROMPT diagnosis and treatment are essential in patients in, or recently returned from, the tropics. But it must not be assumed that all their illnesses are due to exotic diseases. The findings of a thorough general examination must be reviewed with an open mind. While high fever in a European recently entering a malaria endemic area may well be due to malaria a similar fever in an indigenous inhabitant is most unlikely to be due to it, even though some parasites may be found in his blood. Long exposure to malaria infection will result in substantial immunity to the local strains of malaria organisms. Conversely, tuberculosis, the respiratory infections and the exanthemata will cause much more severe illness in natives than in the European, who has greater immunity to them. Furthermore nearly all residents in a given area suffer from malaria which may be just a concurrent and confusing feature of some other illness.

Thick and thin blood films and urine and fæces should be examined as a routine whatever the presenting symptoms and whatever the presumptive diagnosis. Omission of any of them will, sooner or later, make the reason for their routine examination only too clear. An apparently acute surgical abdomen may be due to malignant tertian malaria or amœbic typhlitis. Salpingitis may be bilharzial in origin and cerebral and spinal cord lesions may be caused by schistosomiasis. For all these conditions the appropriate treatment will bring the cure which surgery would not.

The tropical illnesses which may present as medical emergencies include the following:—

- Pernicious malignant tertian malaria.
- Blackwater fever.
- Amœbiasis.
- The helminthiasis.
- Conditions due to heat.

PERNICIOUS MALIGNANT TERTIAN MALARIA

Malignant tertian (*Plasmodium falciparum*) malaria is the only one of the four malaria infections of man which is prone, often suddenly and without warning, to cause grave manifestations. These may be rapidly fatal unless promptly diagnosed and treated. Essentially they are all due firstly to destruction of many red cells and a reduction in the oxygen-carrying capacity of the remainder, and secondly to plugging of small vessels by numerous parasitised red cells with resulting local ischæmia and anoxia.

The complications, on clinical grounds, have been divided into various types.

Cerebral.—This, the most grave immediate complication, may develop over a period of hours. There is increasing headache, drowsiness, and mild or maniacal delirium, coma and death. Parasites, many of them segmenting, are numerous in the circulating blood. Parasitised cells block the capillaries of the brain (Fig. 30).

Abdominal.—These include **bilious remittent fever**, with nausea, vomiting and diarrhœa; enlargement and tenderness of the liver, with jaundice and oliguria—the urine containing albumin, bilirubin and casts. In **dysenteric malaria** there is diarrhœa with the passage of much blood and mucus, and many cells. **Choleraic malaria** is so named because of the copious watery stools with much nausea and vomiting, leading to dehydration with muscular cramps. In all these forms there is a heavy parasitæmia, usually with the presence of segmenting parasites, in the peripheral blood.

Algid.—There is profound prostration with vomiting and diarrhœa; the temperature is high but the skin feels cold and clammy from peripheral circulatory failure which may be fatal. Large numbers of parasites are found in the blood.

Hyperpyrexial.—This commonly occurs as a direct complication of cerebral malaria. The patient's skin is hot and dry and the extremities cyanosed. Delirium rapidly goes on to coma with urinary and fæcal incontinence, and finally to death. The blood contains large numbers of parasites.

DIAGNOSIS.—In malarious countries (see map on page 369) search for a malaria infection is the first step in the differential diagnosis of nearly every acute illness. The blood must invariably be examined. If this is not possible immediately slides should at

least be taken and where the indications suggest pernicious malaria treatment for it should be given without any delay. Pernicious



FIG. 30

Micro-photograph of brain section from a fatal case of malignant tertian malaria. The capillaries are distended and obstructed by parasitised red cells. The heavy black dots in the vessels represent malarial pigment. $\times 400$.

malaria is always associated with the presence of numerous parasites in the blood.

Treatment.—Quinine, chloroquine (Nivaquine) or mepacrine may be used in this order of preference. Proguanil (Paludrine) is *not* suitable for these emergencies as its action is too slow.

Quinine bihydrochloride 0·5 G. ($7\frac{1}{2}$ grains) is given intravenously at once *slowly* and preferably in 284 ml. ($\frac{1}{2}$ pint) of dextrose saline. The dose is repeated six-hourly until the patient is able to swallow, retain and absorb antimalarials given orally; normally this should be after at most three injections. Chloroquine sulphate (Nivaquine) may similarly be given intravenously in a dose of 0·2 G. of the base. Mepacrine is not given intravenously; if given intramuscularly it may cause abscesses and its action is slow.

When the patient has sufficiently recovered to swallow, oral treatment with quinine (2 G. (gr. 30) daily for 3 days), or with chloroquine (1 G. daily for 3 days) or with mepacrine (0·6 G. daily for 5 days) is begun. Chloroquine or mepacrine are safer than quinine for this continuation treatment in view of the tendency of the latter to cause hæmoglobinuria.

Finally, a course of Paludrine (proguanil) (0·6 G. daily for 3 days and then 0·3 G. daily for 7 days) is given to sterilize the infection; this drug so given will almost invariably eradicate a *Plasmodium falciparum* malaria infection.

In addition, collateral treatment of complications may be necessary. A copious fluid intake by mouth should be encouraged. If the red cell count falls below 2 million cells per cu.mm. carefully cross-matched blood should be transfused. The presence of shock indicates a need for the infusion of plasma and other suitable fluids (e.g., dextran) to restore the blood volume. Salt and water loss from vomiting and diarrhoea must be made good. Hyperpyrexia is controlled by fanning a moist (not wet) sheet placed over the body, and thus cooling it until the rectal temperature has fallen to 102°F. Lumbar puncture, by lowering intracranial tension, may relieve a comatose patient.

BLACKWATER FEVER

Massive acute hæmolysis of circulating red cells may take place quite unexpectedly in those who have for some time suffered from malignant tertian malaria. It is particularly prone to occur in those who have been taking prophylactic drugs, especially quinine, irregularly or in inadequate dosage. The attack may occur spon-

taneously or may follow a dose of quinine. It may be an isolated event and last only a few minutes, or it may recur at short intervals over a period of days.

The clinical manifestations of each hæmolytic episode are a rigor, nausea and vomiting, fever, pains in the loins, and immediately thereafter the passage of blood pigments in the urine—hence the name. Jaundice develops in proportion to the hæmolysis, which obviously will cause a sudden profound anæmia. The pigments found in the blood plasma are oxyhæmoglobin and methæmalbumin; the latter is formed from oxyhæmoglobin, which is removed from the plasma in this form by the reticulo-endothelial system. The pigments passed in the urine are oxyhæmoglobin and methæmoglobin, the former changing into the latter in an acid urine. The urine is also loaded with albumin, renal epithelium and casts, and amorphous debris; its reaction usually, though not invariably, is acid, and its volume is reduced.

In severe cases there may be anuria. Uræmia following this is one of the principal causes of death from blackwater. The suppression of renal secretion is now considered to be due to cortical ischæmia following the operation of the circulatory shunt mechanism in the kidneys, and so is comparable to that occurring in other conditions associated with vascular collapse. When the urinary output increases after the end of the attack of blackwater an increased volume of a low specific gravity urine is passed for a time; no permanent kidney damage follows.

Another immediate cause of death is the severe anæmia occasioned by the massive hæmolysis. All degrees of severity of hæmolysis are seen; in the most severe the red-cell count may fall within 24 to 48 hours even to below one million cells per cu.mm. of blood. Hyperpyrexia is yet a further complication of blackwater which may cause death.

DIAGNOSIS.—Hæmoglobinuria occurring in those resident in, or recently returned from, a malignant tertian malaria endemic area should always be suspect as blackwater fever. When this is sudden, marked, and repeated, and is associated with rigors, fever, and a rapidly developing anæmia with jaundice, the clinical diagnosis is clear. Some care must be taken to avoid confusion with passage of a dark-coloured urine or with hæmaturia; microscopical examination of the urinary sediment for red cells and a spectroscopic search for the presence of blood pigments are very desirable to put the matter beyond doubt.

Malaria parasites are only rarely present in the blood after an attack of blackwater fever has started; therefore examination of the blood for them does not help in confirmation of the diagnosis.

Treatment.—When blackwater develops all unnecessary movement of the patient and needless transportation must be avoided. Absolute rest in bed is essential. The patient should be encouraged to drink freely, and he must at once be put on a properly kept fluid chart. Every specimen of urine passed must be kept for inspection, and its volume, colour and reaction noted. The temperature and pulse rate should be recorded two-hourly.

Unless parasites are evident in the blood no antimalarial drugs should be given over the period that the blackwater persists. If parasites are found chloroquine (Nivaquine) (0.5 G. daily for 3 days) or mepacrine (0.3 G. daily for 5 days) should be given, *but never quinine*. Forty-eight hours after the blackwater stops paludrine (0.6 G. daily for 3 days and then 0.3 G. daily for 7 days) must always be given to sterilize the malignant tertian malaria infection, which otherwise will relapse within a week or two.

Peripheral circulatory failure is treated by restoring the blood volume by the infusion of plasma, dextran, saline and glucose. The latter should be given early when persistent vomiting prevents an adequate intake of fluid orally, and sodium bicarbonate should be given to remedy the salt loss and adjust ionic imbalance. When the red-cell count falls below 2 million and there is no evidence of the blackwater stopping, blood transfusion becomes necessary. The cells and the sera of both donor and recipient must be cross-matched immediately before each transfusion. The red cells of a healthy donor are hæmolyzed after introduction equally as readily as are those of the recipient blackwater fever patient during a hæmolytic episode; large volumes of blood, therefore, should not be introduced at a time. When transfusion becomes necessary it is better to give repeated pint (568 ml.) transfusions, rather than a larger transfusion on a single occasion. For the same reason, and also because transfusion might of itself precipitate a further intravascular hæmolysis, transfusion should not be undertaken lightly. Once the hæmolysis has ended the patient, if provided with iron, will rapidly make good his loss.

After an attack of blackwater fever exertion should be avoided until the patient has recovered from the severe anæmia and other immediate consequences of the attack. In addition to symptomatic treatment during convalescence, the important thing to remember

is that the causative malignant tertian malaria infection must be eradicated without delay, or relapse inevitably will soon occur. Paludrine is the drug of choice for this purpose. Convalescence is usually quite speedy and recovery complete.

AMŒBIASIS

Infection of the human large bowel with *Entamoeba histolytica* may be symptomless or it may cause relapsing amœbic dysentery. Extra-intestinal spread may occur locally or may extend to the liver by emboli. From the liver the infection, if neglected, spreads directly to neighbouring structures, and it may again spread embolically to other sites in the body, such as the spleen or the brain.

Hepatic amœbiasis. This is the result of lodgment in the liver of amœbæ which have eroded their way into radicles of the portal

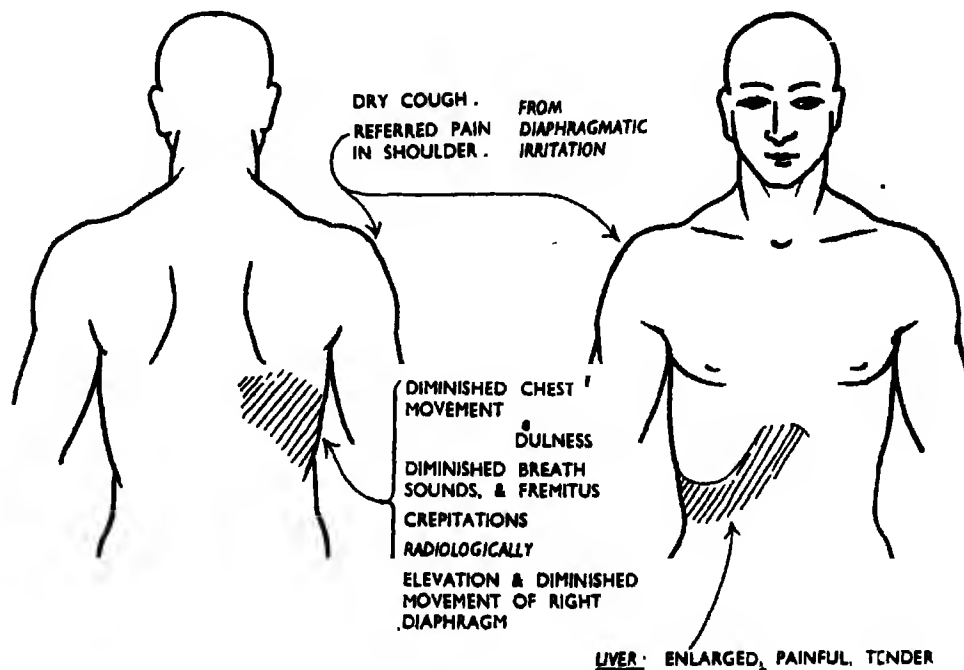


FIG. 31

Amœbic infection of liver.

vein in the wall of the large intestine. It may develop in apparently symptomless carriers but much more commonly it does so in patients with a history of relapsing amœbic dysentery. The individual amœbæ give rise to small colonies of multiplying amœbæ which form small bacteriologically sterile abscesses consisting of lysed liver tissue around the margins of which each

colony thrives. These foci are usually multiple, and the condition at this stage is referred to as amoebic hepatitis.

The onset of hepatitis is usually insidious but may be acute; it does not develop during an acute attack of dysentery but occurs during a quiescent phase. There is slight irregular fever, discomfort and fullness over the liver area, with tenderness and some enlargement of the liver due to engorgement. The individual lesions enlarge and coalesce to form an irregular amoebic abscess. It commonly takes a period of a few weeks to reach this stage, but it may develop acutely within a week or two of the establishment of the liver infection.



FIG. 32

A liver abscess causing deformity and elevation of the right dome of the diaphragm.

(Dr. Carmichael Low, by courtesy of Sir P. Manson-Bahr.)

The manifestations of an amoebic liver abscess are enlargement of the affected lobe of the liver, commonly the right, local discomfort, tenderness and often pain referred to one shoulder or the other according to the area of liver and adjacent diaphragm affected (Fig. 31). There is irregular fever, often with drenching sweats and progressive loss of weight and of condition. The patient is prostrated and his complexion becomes muddy and sallow. There

is no jaundice. There is a leucocytosis, due to neutrophil increase, up to possibly 25,000 cells per cu.mm. of blood. The liver enlargement is around the lesion, and so may be apparent upwards or downwards or outwards from the affected lobe according to the site of the infection of the lobe. As the upper part of the right lobe of the liver is the usual site of the infection, so the right diaphragm usually is found to be raised and irregular and its movement restricted when examined radiologically (Fig. 32).

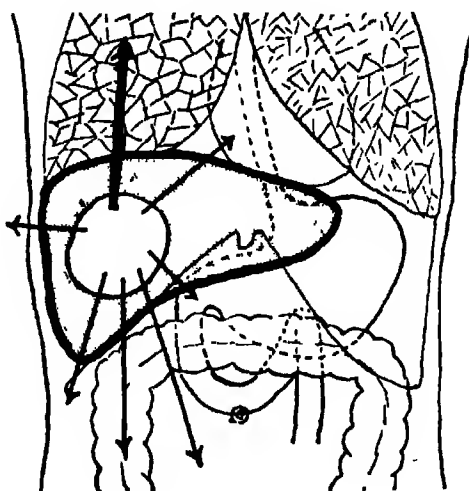


FIG 33
Directions in which a tropical
liver abscess may burst.
(After Cope.)

When the infection reaches the surface of the liver it extends beyond it to neighbouring tissues, commonly the diaphragm. The amœbæ infect this and extend through it to the pleura and the lung. There is now pulmonary amœbiasis, with physical signs of the lung involvement. The lung tissue is lysed by the parasites; if a bronchus is eroded lysed lung and liver tissue containing amœbæ is expectorated. This must not be confused with hæmoptysis. From these extensions amœbæ may further be conveyed embolically; the rare amœbic brain abscess is an example of such further embolic spread.

Direct extension, instead of taking place through the diaphragm, sometimes occurs through the chest or abdominal wall, and then the abscess drains spontaneously to the exterior. Rupture may occur into the peritoneum or abdominal organs, usually with a fatal result (Fig. 33).

An occasional complication of an amœbic liver abscess is secondary bacterial infection. When this occurs the fever becomes

greater and more continuous, the leucocytosis mounts sharply and the condition of the patient very rapidly deteriorates.

Atypical intestinal amœbiasis.—This may present as an emergency in two forms:—

Amœbic typhilitis.—This, because it causes pain, tenderness and thickening in the right iliac fossa, resembles appendicitis but the symptoms are usually less urgent. Vegetative *E. histolytica* will be found in the stools. When operation is not absolutely imperative it should be deferred until the effect of daily injections of emetine has been observed.

Amœboma.—An amœbic granuloma may develop in the abdomen with signs of intestinal obstruction and so may suggest a neoplasm. Whenever there is a possibility of amœbiasis in a patient with a tumour of the bowel, emetine should be given in addition to any other treatment that may urgently be indicated. A history of residence in the tropics even many years previously, and of acute attacks of amœbic dysentery, even though not of recent occurrence, in conjunction with the symptomatology and physical findings should always suggest this diagnosis. The stools should be searched daily for proof of the original infection. If this is established it helps to confirm the diagnosis of amœboma. If parasites are not found this does not necessarily exclude it, as sometimes they are very scanty when secondary amœbic spread has occurred and can only be recognized after repeated stool examinations.

Treatment.—Emetine hydrochloride, 65 mg. (gr. 1) daily intramuscularly for 12 days, will almost invariably rapidly sterilize the amœbic infection in the liver, or any other extra-intestinal focus of amœbic infection. If the liver destruction is slight the damaged tissue will be absorbed; if it is very considerable it may be necessary to remove it by aspiration, or even by surgical drainage if its location and extension indicate a need for this. Under no circumstances must the specific treatment of the amœbic infection be omitted, or the lesion will continue to extend however effective the drainage. There is no point in introducing emetine into the abscess cavity at the time of aspiration or drainage.

If emetine is contra-indicated by heart disease or idiosyncrasy, the anti-malarial drug chloroquine affords a most useful and effective alternative to emetine in the treatment of hepatic

amœbiasis. The dose is 1 G. daily by mouth for 5 days to one week. It may be given synchronously with emetine treatment.

When the extra-intestinal amœbic infection has been controlled and cleared the primary intestinal infection will still remain and must be eradicated, or further extra-intestinal spread may occur. The sterilisation of the bowel infection entails the use of a number of amœbicidal drugs given in concert over a period, and among these emetine in some form, but not chloroquine (Nivaquine) is included.

THE HELMINTHIASES

Some urgent situations may arise from infestation of the bowel or tissues with certain worms.

Acute allergic conditions.—These may result from accidents such as the rupture of a hydatid cyst. The migration of many larval schistosomes through the skin and tissues immediately after infection can cause a sharp allergic reaction in those unduly susceptible. This is also liable to occur with even greater frequency and in a more pronounced form some weeks later, when the adult worms, having developed in the liver, migrate up the portal vein and its tributaries to their elective habitat.

Many of the pathogenic filarial worms cause allergic manifestations, probably due solely to the adult worms and not to the microfilariae they produce. *Loa loa* gives rise to sudden "calabar swellings" which last a few days. *Wuchereria bancrofti* causes a bizarrely situated retrograde-type lymphangitis with an associated lymphadenitis, and often a general systemic reaction (filarial fever). In either of these two cases a search for microfilariae in the blood should be made; for *Loa loa* this is done during the daylight hours, and for *W. bancrofti* nocturnally. Microfilariae may not be recoverable from the peripheral blood until patients have suffered from symptoms for a year or two. Several of the filarial infections of man may cause a severe and long-continuing irritant dermatitis of a toxic type.

Acute mechanical complications.—These may result from the presence of worms both in transit during migration and when in their normal habitat, or they may follow abnormal migrations. The transit of certain larval nematodes, especially those of round worms (*Ascaris lumbricoides*), in large numbers through the lungs during initial infection may cause an acute verminous pneumonitis with fever, prostration and cough with the expectoration of

blood-tinged sputum containing the larvæ. Round worms are from 7 to 9 inches long, and if there are some dozens of them, as is often the case in children, they may become impacted in the gut or cause perforation, volvulus or intussusception. When individual worms ascend into the stomach they may remain there and cause intractable vomiting until the worm is expelled. Occasional worms ascend the œsophagus and cause laryngeal spasm, and they may pass up the posterior naso-pharynx and lodge in, or emerge from, the nose. Individual ascarids on rare occasions may ascend the pancreatic or common bile duct, and may rupture these, causing an acute surgical emergency.

The adults of the very common West African filarial worm, *Loa loa*, wander beneath the skin and mucous membranes. Often they travel beneath the conjunctiva causing acute discomfort and alarm but no permanent damage. When clearly seen there they can be seized with fine forceps. The conjunctiva at the point of hold is then snipped and the worm withdrawn intact. The sense of relief the patient experiences is remarkable. Sulphacetamide (Albucid) 10 per cent. or some similar drops are applied to the conjunctiva for a day or two and the patient given a course of diethylcarbamazine (Banocide; Hetrazan) to kill the other adult worms he probably harbours.

Tapeworms.—Infestations with adult cestodes are diagnosed by the detection of gravid segments of the worms voided from the bowel. The species of worm can be determined by study of the anatomy of the segment.

Tænia saginata (Fig. 34), the beef tape-worm, is a harmless though æsthetically unpleasant parasite acquired by consumption of the living larvæ (*cysticercus bovis*) which develop only in bovines. The latter acquire their larval infestation by swallowing the eggs contained in the gravid segments voided by man.

Tænia solium (Fig. 35), the pork tape-worm, is equally harmless in so far as the adult worm is concerned; but unfortunately man himself as well as the pig can act as intermediate host for the development of the larvæ. So if man swallows the eggs formed by his own or another's parasite he develops larvæ (*cysticercus cellulosæ*) in his muscles and other tissues, including the central nervous system, and suffers from cysticercosis, a very grave and often fatal condition. In addition to swallowing eggs, it has been postulated that infection of man with cystercerci may follow infestation with an

adult worm when a segment is regurgitated up the bowel by retro-peristalsis; in this case, as each gravid segment contains some thousands of eggs, the infection is an extremely heavy one.

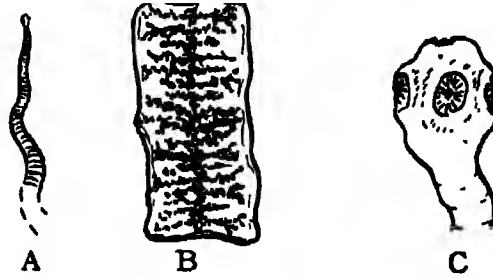


FIG. 34

A and B Head and *mature* segment of *T. saginata*. Natural size. Note 16 or more lateral uterine branches (c.f. *T. solium*). Mature segments are longer than broad. C. Head of *T. saginata* $\times 20$.

It therefore follows that the discovery of passage of tape-worm segments constitutes an emergency until the segments have been proved to be those of the harmless *T. saginata* and not those of the potentially highly dangerous *T. solium*. If the worm proves to be the latter every precaution must be taken to ensure that neither

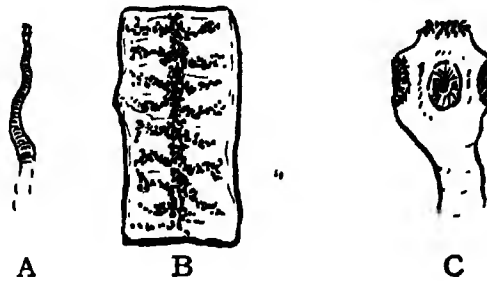


FIG. 35

A and B. Head and *mature* segment of *T. solium*. Natural size. Note only 8-12 lateral uterine branches. C. Head of *T. solium* $\times 20$. Note double row of hooklets.

the patient himself nor anyone else can swallow the innumerable eggs contained in the gravid segments. Furthermore, the patient must be relieved of his parasite at the earliest possible moment, and for some years thereafter the possibility of his having cysticercosis must be envisaged. The cysticerci begin to die two or three years after formation, and the majority are dead within five years. Those in muscles calcify, but those in the central nervous system

do not. Dead and calcified cysticerci when seen radiologically in the muscles afford an inferential diagnosis of cerebral cysticercosis.

Treatment.—First the bowel is cleared of obscuring solid matter; second, the anthelmintic is given; and third, the worm and the anthelmintic used must be expelled by sharp purgation.

The drug traditionally used is filix mas. The patient is given nothing but fluids for 48 hours, and then a saline purge (magnesium sulphate 15 G. ($\frac{1}{2}$ oz.)). After this has acted three doses, each of 1.7 ml. (m 30) of filix mas are given at half-hourly intervals and are followed two hours later with 30 G. (1 oz.) of magnesium sulphate. All stools passed in the ensuing 48 hours are searched for the head.

A more effective treatment is as follows. After 48 hours' starvation 200 mg. (gr. 3) of sodium amytal are given and a Ryle's tube is passed at night. Next morning, when the tube will have travelled well beyond the pylorus, 1 G. of mepacrine suspended in 100 ml. of warm water is introduced through the tube with a syringe; half an hour later 45 to 60 G. ($1\frac{1}{2}$ to 2 oz.) of magnesium sulphate are likewise introduced in another 100 ml. of warm water, and the tube is withdrawn. A warm drink is then given and within an hour or two the parasite usually is passed intact, tightly contracted and stained yellow.

Passage of the body of the worm, short of the head, is of no benefit for the worm will grow again within three months. If the head is not recovered immediate further treatment is likely to be ineffective and may be dangerous. The return of segments in the stools should be awaited.

CONDITIONS DUE TO HEAT

Environmental conditions which cause interference with the heat-controlling mechanism of the body, and disturbances in the electrolyte-water balance and of the cardiovascular system, lead to various forms of acute physiological breakdown. The clinical manifestations of these abnormalities, either singly or in combination, lead to the following clinical conditions:—

- (1) Heat pyrexia and hyperpyrexia.
- (2) Thermogenic anidrosis.
- (3) Heat exhaustion.

Heat pyrexia and hyperpyrexia.—This is caused by exposure to intense heat over a period of at least some hours, and usually of days or weeks. It occurs more readily in the unacclimatized than in the acclimatized and is primarily due to a complete failure of sweating. Frequently it is complicated by electrolyte-water imbalance and by shock. It may be precipitated by over-vigorous muscular exercise, by an unduly high protein diet with resultant increased metabolism, or by endocrine hyperactivity, particularly of the thyroid gland.

Usually there are prodromal symptoms for several days; occasionally the onset is quite unexpected and sudden—the so-called “flash hyperpyrexia.” The development is usually fairly rapid; the patient first suffers from headache, restlessness and mental confusion, often with nausea and vomiting; commonly there is severe thirst and the resultant heavy fluid consumption causes polyuria. The skin feels hot and it becomes quite dry and flushed as sweating stops; the pulse and respiration rates rise. Mental changes, delirium and coma rapidly appear, and there may be convulsions. The temperature rapidly mounts, and it continues to rise until it exceeds 110°F. before death.

Medical shock due to vascular collapse may occur at any stage, and unless promptly treated can cause irreversible changes in the brain, liver and kidneys. The onset of shock is marked by a rapid fall in temperature, a fall in the circulating blood volume and a rising hæmoconcentration, with a fall in the blood pressure. There is oliguria going on to anuria, which is followed by death from uræmia. Evidence of dehydration and of salt loss are usual when there has been severe vomiting and diarrhœa. But these latter conditions are only secondary to the primary heat hyperpyrexia, which is due to failure of sweat secretion.

Treatment.—The aims are to lower the body temperature and to restore sweating. These are achieved by cooling the body, by evaporating water from its surface by fanning or blowing air on to a damp sheet placed over it. This should go on until the rectal temperature falls to 102°F., when natural sweating is usually restored. If it is not, and the temperature rises again, the treatment is repeated as necessary.

Shock and dehydration, if present, are treated by the parenteral introduction of suitable fluids, a close watch being kept on the fluid balance.

Thermogenic anidrosis.—This occurs after long exposure to hot conditions. It is due to a partial failure of sweating, chiefly over the trunk and limbs. The face and neck continue to sweat and there is no hyperpyrexia. The concentration of salt in the sweat secreted is high (0·5 per cent. or more) and on this account the condition has been ascribed to fatigue of the sweat glands. There are hyperkeratotic changes in the non-sweating skin. Cardio-vascular failure and electrolyte-water imbalance may be secondary complications.

The onset usually is slow over a period and is associated with increasing fatigue, dyspnoea and palpitation after exercise, chiefly during the hottest part of the day. The temperature rises to 100° or 101°F.; there is profuse sweating on the face and neck, only slight sweating on the palms and axillæ, feet and groins, and none on the trunk and limbs. There is a patchy mamillaria of the dry parts of the skin and there may be œdema of the fingers and arms. There will be polyuria with a low specific gravity urine if the patient drinks much, but the gross urinary salt output usually is normal.

Treatment.—Rest in cool well-ventilated surroundings for some days and the oral administration of salt and water in adequate amount is usually all that is necessary.

Heat exhaustion.—This is most commonly seen as a sequel to manual labour in hot humid surroundings. It is due to electrolyte-water imbalance complicated by cardiovascular disturbance. There is no failure of sweating. Heat exhaustion may be precipitated by any co-existing febrile illness.

The onset is usually insidious over some days, but it may be abrupt. There is increasing headache, nausea and vomiting, and fleeting muscular cramps. The urine is scanty but unconcentrated; it contains albumin and casts, and its chloride content is low or nil (Fantus' test, *page 463*). In severe cases there is circulatory failure, with reduction in the circulating blood volume, hæmoconcentration, and a low blood pressure. There may be suppression of urine if shock is marked.

Treatment.—This consists essentially of restoration of the electrolyte-water balance and the blood volume. Five to six pints of fluid and 25 to 40 G. of salt should be given orally in the first 24 hours to mild cases. In more severe cases, with marked dehydration and shock, plasma and then fluid and salt must be

given parenterally. The initial intravenous infusion should be one pint of plasma over 20 to 30 minutes, and then one pint of physiological saline in the next 15 minutes, and another in the next half-hour. A total of six pints of fluid intravenously should not be exceeded in the first 24 hours. The response is usually rapid and the patient may be fully convalescent within a couple of days.

A. R. D. ADAMS.

CHAPTER XIX

Emergencies in Industrial Medicine

THE medical hazards of industry are numerous and may be classified as follows:—

PHYSICAL.

Temperature.	Heat cramps	(page 340)
	Heat exhaustion	(page 340)
	Heat stroke	(page 339)
Pressure.	Compressed air illness	(page 375)
Electric shock.		(page 118)

CHEMICAL.

- (1) **Asphyxiants**, causing oxygen deficiency without direct injury to respiratory tract:
- (a) simple, inert gases displacing oxygen from the respiratory tract, *e.g.*, nitrogen, carbon dioxide, nitrous oxide.
 - (b) chemical, interfering with the vascular respiratory mechanism, *e.g.*, carbon monoxide, hydrogen cyanide and allied products; aniline, nitrobenzene and other nitro- and amido-derivates of benzene.
- (2) **Irritants**, causing inflammation of respiratory tract and lungs:
- (a) acting on upper respiratory tract, *e.g.*, ammonia, hydrochloric acid and sulphur dioxide.
 - (b) acting on upper respiratory tract and lungs, *e.g.*, sulphur dioxide, chlorine and other halogens, hydrofluoric acid.
 - (c) acting principally on lungs, *e.g.*, nitrous fumes, phosgene, arsenic trichloride, nickel carbonyl.

- (3) **Centrally acting poisons** Volatile solvents—
 producing anæsthetic effects after absorption through the lungs:
- (a) hydrocarbon group, *e.g.*, benzene, toluene, petrol, benzine, white spirit, and solvent naphthas.
 - (b) chloro-compounds, *e.g.*, trichlorethylene, carbon tetrachloride, methylene dichloride, methyl chloride, methyl bromide, chloroform.
 - (c) alcohols, *e.g.*, methyl alcohol.
 - (d) miscellaneous, *e.g.*, organic nitrogen compounds acting upon the blood and circulation ("nitrite effect"); amyl nitrite.
- (4) **Miscellaneous poisons:** Lead and tetra-ethyl lead; arseniuretted hydrogen; hydrogen sulphide; carbon bisulphide, nicotine; phenol; metallic particles producing metal fume fever.

(For poisoning on board ship see page 373)

Poisonous substances may enter the body in three ways:—

- (1) **VIA THE LUNGS.** This is by far the most important route. Inorganic dusts, the fumes or dusts of metals, the vapours of organic solvents, and most toxic gases are absorbed in this way.
- (2) **VIA THE SKIN.** This route is less common, but important because of the need to remove contaminated clothing promptly. The noxious substances must be removed by washing or other means. Tetra-ethyl lead, aniline, nitrobenzene, phenol, cyanide and insecticides can be readily absorbed through the skin and rapid poisoning result. It must

also be remembered that liquids splashed on the clothes (*e.g.*, liquid phosgene) will vaporise when the patient is taken indoors, and aggravate his symptoms by being inhaled.

(3) VIA THE GASTRO- INTESTINAL TRACT.

This is comparatively unimportant in industry, but applies to accidents in laboratories.

If a person is taken suddenly ill while at work, it is usually possible to obtain a detailed history of his precise occupation, and to assess the results of exposure to toxic substances. Works managers, chemists and engineers are becoming increasingly aware of the symptoms of industrial poisoning, but it must be emphasised that accurate information regarding the patient's occupation is indispensable in establishing the correct diagnosis. It is essential to know the materials to which the patient has been exposed, and if the patient is seen away from his place of work (and many cases of occupational poisoning arise some hours after leaving work), a direct approach should be made to the factory concerned. In doubtful cases the help of the Medical Inspector of Factories should be sought.

The common emergencies arising from occupational poisoning are **asphyxia**, **coma** (*see page 189*), and **convulsions** (*see page 203*). Less serious symptoms are pulmonary irritation and a variety of symptoms arising from interference with the functions of the central nervous system. A few industrial poisons produce characteristic clinical pictures, 'but, in the main, the symptoms do not differ from those which may arise from acute illness of non-occupational origin. Even with a complete history and details of exposure, non-industrial illness has first to be excluded. The principles of treatment are well established and few cases of occupational poisoning require specific antidotes.

GENERAL PRINCIPLES OF TREATMENT.—On receiving an emergency call the doctor will usually find that the first indication is for the symptomatic treatment of asphyxia and coma. First-aid measures will have been adopted as a rule efficiently, because of the increasing attention given to this service in industry.

Note on gassing casualties sent to hospital.

A medical panel of the Association of British Chemical Manufacturers has drawn up a series of labels which can be attached to gassing casualties before they are sent to hospital. These labels

show what first aid has been given and indicate what immediate treatment is recommended.

ASPHYXIA

Asphyxia may be assumed to be present in all cases showing cyanosis, as well as in all cases of carbon-monoxide poisoning. When coma is the presenting sign there is almost invariably some degree of asphyxia and death results from respiratory failure. •

RESCUE.—When called to casualties after an escape of gas the following principles of rescue must be observed.

- (1) Keep to the windward side of the gas leakage and warn people in the vicinity.
- (2) Put on a breathing apparatus (*page 541*).
- (3) Shut off the source of gas, if this is possible.
- (4) Open all doors and windows and leave the building until it is clear of gas.

After rescue, it is essential to keep the patient at rest, even if he is only slightly affected, and no exercise must be permitted for some hours after recovery. After removal of the patient from contact with the poisonous material, which may include stripping and washing him, treatment should be as follows.

Treatment.—Remove the patient to a pure atmosphere and lay him down facing the wind. Don't walk him about under any circumstances.

- (1) Ensure and maintain an adequate airway.
- (2) Commence artificial respiration by the Holger Nielsen method (*page 544*). Where special risks obtain, apparatus is commonly provided for Eve's rocking method. The doctor should stress that success depends largely on prompt action. Many cases of industrial poisoning require very prolonged artificial respiration.
- (3) Remove or cut away contaminated clothing (*e.g.*, splashed with HCN).
- (4) Keep the patient warm with blankets and hot-water bottles.
- (5) Administer oxygen by a portable apparatus ("The Novox," Siebe, Gorman & Co., Ltd. [*page 559*], The Oxford Inflator [*page 561*], or a face mask [*page 573*]). This is unquestionably the most important single measure in the treatment of acute medical emergencies in industry.

- (6) Circulatory failure may call for use of analeptics (*see page 608*). It is almost invariably secondary to asphyxia, however, and its effective treatment depends upon the relief of the asphyxia as already described.

Venesection should not be practised for cyanosis alone, but only if there is evidence of embarrassment of the right side of the heart, such as marked engorgement or pulsation of the jugular veins.

Having dealt with the immediate emergency measures, more detailed methods of dealing with individual poisons may be considered.

ASPHYXIANTS

All users of asphyxiant products must be acquainted with the first-aid measures required in the event of an accident, and resuscitation and rescue apparatus should always be at hand. Workers should be practised in the use of the equipment and in methods of artificial respiration. The action of many of the compounds in this group (HCN in particular), in high concentrations, is so sudden that a man entering a contaminated atmosphere may collapse before he has time to act on any warning he may receive.

Simple asphyxiants cause asphyxia by oxygen deficiency but do no direct injury to the respiratory tract. Carbon-dioxide is a good example and its effects are encountered in a variety of places, *e.g.*, breweries and mineral water works, lime kilns and ships' holds.

Of the chemical asphyxiants, **carbon monoxide** is the most important and is responsible for between one half and one third of all reported gassing accidents in factories in Great Britain. The risk exists in many industries, particularly in those with gas-producers and blast furnaces, but exposure to coal gas and motor exhaust fumes occurs in all spheres of life. Having no colour, vapour or odour, CO is rapidly absorbed via the lungs, and is a particularly dangerous substance. Entry into high concentrations produces almost instantaneous loss of consciousness. For treatment *see page 7*.

Cyanides are encountered in many chemical industries and are used in dyeing, the manufacture of plastics, for fumigation and for the hardening of metals.

Hydrogen cyanide is highly toxic, colourless and volatile, having an odour of bitter almonds, which may not be noticeable however, even in high concentrations. It probably acts directly on the body cells, interfering with normal oxidation. The respiratory centre is particularly sensitive to its action, which accounts for the early respiratory paralysis.

In industry, poisoning results rarely from swallowing solid or liquid cyanide. It more commonly arises from the inhalation of hydrocyanic acid gas (HCN).

Owing to the high volatility of HCN the danger of absorption after spilling the liquid on the bare skin appears to be slight, as long as evaporation is unimpeded.

The action of HCN in high concentrations is so sudden that a man entering an atmosphere containing it may collapse at once. It is important, therefore, that when the presence of HCN in the atmosphere is suspected, a benzidine-copper acetate test paper should be exposed before the room is entered. It is prepared by saturating a strip of absorbent paper with a solution freshly made by mixing equal parts of an aqueous solution containing three grammes of copper acetate per litre and an aqueous solution containing one gramme of benzidine acetate per litre. This is exposed for at least 10 seconds. If HCN is present it turns blue.

SYMPTOMS OF POISONING.—Early warning symptoms are irritation in the throat, increasing difficulty in breathing, lachrymation, followed by headache, dizziness, nausea, vomiting and general weakness with a feeling of heaviness in the arms and legs.

At the first sign of any of these symptoms a person must immediately leave the dangerous atmosphere. Warning notices should be immediately affixed in the area and the test for HCN carried out. Thereafter entry is at the discretion of a responsible member of the technical staff, who will naturally supervise all further investigations and safety measures.

Signs of severe poisoning are pallor and increasing shock, unconsciousness, and cessation of breathing. There may be tetany and convulsions, and signs of cardiac failure.

FIRST AID MEASURES.—When the patient has been rescued and the contaminated clothing removed or cut away, break a capsule of amyl nitrite, and allow the patient to inhale the vapour. Immediately proceed with artificial respiration and the adminis-

tration of oxygen. If cyanide comes into contact with the skin the affected area should be repeatedly washed with water.

Treatment.—The modern treatment of cyanide poisoning is based on the administration of nitrites, which convert cyanide in the blood into the non-toxic cyanmethæmoglobin. It is believed that the nitrite combines with some of the available hæmoglobin to form methæmoglobin, which then combines with cyanide to form cyanmethæmoglobin. Amyl nitrite is readily absorbed so long as the patient is breathing, and is stated to form methæmoglobin in 10 to 15 seconds.

Following rescue and first aid measures, the following treatment is advised:—

- (1) Continue with artificial respiration, if possible using Eve's rocking method.
- (2) Administer analeptics (*see page 608*).
- (3) Repeat amyl nitrite inhalations for 3 to 5 minutes.
- (4) Inject a solution of sodium nitrite, 0·3 G. in 10 ml. of water intravenously at a rate of 2 to 5 ml. per minute. (This should not be kept in solution unless an anti-oxidant like sodium sulphite is present, but it can be kept dry in ampoules).
- (5) Inject intravenously through the same needle, 25 to 50 ml. of a 50 per cent. solution of sodium thiosulphate at the same rate. This increases the efficacy of the treatment by converting cyanmethæmoglobin into a relatively harmless and easily excreted thiocyanate.

These procedures may be repeated, in half dose, an hour later if symptoms recur. It is necessary to observe the patient closely for 24 to 48 hours.

Aniline and the nitro and amino derivatives of benzene.

These compounds cause pallor and cyanosis by the formation of methæmoglobin. The grey-blue colour of the lips is characteristic. Asphyxia and nervous symptoms such as headache, vertigo, nausea and vomiting are common. There may also be irritability, somnolence, or mental confusion, unsteady gait, muscular tremors, and finally convulsions and coma. It is especially important to remember that absorption takes place through the skin, as well as by the lungs, and that the patient may become affected at home, or on his way home. He should be immediately stripped and washed and have a complete change of clothing. Otherwise treatment is symptomatic.

Mono-nitrobenzene (mirbane oil), which is used for making shoe and floor polish, causes similar but less acute symptoms to dinitrobenzene. Similar effects are produced by the nitrophenols. Treatment is along general lines and no specific remedies are indicated.

LUNG IRRITANTS

In many industrial processes exposure to lung irritant gases or vapours may occur. Some of these act on the upper respiratory tract causing immediate irritative symptoms, while others have a lesser effect there but damage the lungs and cause serious delayed symptoms. There is little essential difference between the actions of the various members of this group, and the treatment and prognosis depend on the extent of the lesions in the lungs. The concentration of gas to which the patient is exposed largely determines the clinical picture.

Hydrochloric and sulphuric acids and ammonia cause severe burns, although intense irritation of the upper respiratory tract and eyes may occasionally occur without obvious burns. Bronchitis may ensue, but serious complications such as pulmonary oedema are rare.

Chlorine causes immediate irritation of the upper respiratory and lachrymatory tracts and also considerable bronchial spasm and discomfort. Patients are disabled immediately, but recover quickly and the only after effects are laryngitis and bronchitis. In mild cases, the face is flushed, the respiratory rate slightly increased, and the cough painful; in more severe cases there is considerable respiratory embarrassment with distended veins and cyanosis. Medium rhonchi and râles are heard in the chest.

Sulphur dioxide produces effects in chemical and furnace workers similar to those of chlorine.

Phosgene, used in the dyestuffs industry, **nitrous fumes**, encountered in the manufacture of nitric acid and explosives and in welding, and **nickel carbonyl** all have important delayed effects on the lungs. The initial symptoms are less severe than those caused by irritants previously mentioned. The patient recovers fairly rapidly, and may not appear distressed or ill, but delayed pulmonary oedema may occur. The difficulty in deciding the degree of gassing is notorious, for the condition of the patient immediately after the accident is no guide to the ultimate outcome. Absolute rest should be secured at once and the patient

observed in hospital for 24 to 48 hours in order to prevent pulmonary œdema, or minimise its development.

Beryllium is a metal which is being increasingly used in the field of atomic energy, in copper and other alloys and in radiological equipment.

Exposure particularly to the oxide may cause acute pulmonary symptoms. In the acute disease there is irritation of the eyes and naso-pharynx and a diffuse pneumonitis. Exudates are present in the alveoli and there is marked lymphocytic and fibroblastic reaction. Polymorphs and necrosis are usually absent. The typical lesion is a granuloma of the lungs, skin, liver and lymph nodes. The acute illness is characterised by cough, dyspnœa and cyanosis, which is out of proportion to the pyrexia. Râles are present on auscultation. Complete recovery is likely in two to three weeks but may take two to five months. There is no specific treatment. Convalescence should be prolonged as pulmonary œdema may follow too early activity.

Treatment of lung-irritant gassing.—The two essentials are rest and oxygen. The patient should be carried into a pure warm atmosphere, free from draught. He must not be permitted to walk. Clothing should be loosened and contaminated garments removed. He must be kept at rest, though an occasional change of position from lying down to sitting up may be beneficial. He should be reassured, and encouraged to suppress his desire to cough.

Warmth (hot water bottles and blankets) should be applied, and drinks of hot sweetened tea or coffee given. Dyspnœa or cyanosis should be treated by the administration of oxygen (*see page 569*). When there is pulmonary œdema as shown by diffuse moist sounds and frothy sputum it is sometimes advantageous to give oxygen under pressure during inspiration but a special apparatus is needed (*see page 574*). Artificial respiration is not indicated.

Immediate symptoms are more distressing in cases caused by irritants such as chlorine, which affect the upper respiratory tract. Relief can be obtained by the inhalation of a steamy atmosphere from a bronchitis kettle or from a vessel containing two teaspoonfuls (8 ml.) of Friars' Balsam (Compound Tincture of Benzoin B.P.) in a quart (1 litre approx.) of hot water. Cough should be relieved as suggested on *page 145*. It is desirable to give morphine and little benefit is to be expected from the use of atropine, even in

cases where pulmonary oedema may be expected. Venesection should only be practised if there is definite evidence of embarrassment of the right heart, and not for cyanosis alone. If circulatory failure is evident, Injection of Nikethamide or Leptazol (1 to 2 ml.) may be beneficial.

THE VOLATILE SOLVENTS

Various solvents are used in industry both in large works and in small premises such as dry-cleaning establishments. Many are good anæsthetics and some have delayed toxic effects on the liver and kidneys. Toxic symptoms include headache, giddiness, and a sense of heaviness of the legs, followed by inebriation, drowsiness and loss of consciousness. Death may follow from respiratory failure. Treatment is on general lines—rest, oxygen and artificial respiration.

It is important to recognise certain special features of particular solvents. Many have a heavy vapour and the patient exposed to them must be kept off the floor. Some solvents are inflammable and others such as trichlorethylene are decomposed at red heat, and it is not permissible to smoke in, or near, their vapours. A person affected by solvent vapours, even if only slightly, must in no circumstances be permitted to resume work or exert himself by walking or cycling. He should be taken home by car and instructed to rest for the remainder of the day, preferably in bed.

Benzene, Toluene.

Exposure to small concentrations causes headache, giddiness, muscular twitchings and excitement. After severe exposure these symptoms may be followed by convulsions, coma and death.

Chloro compounds.

Methyl chloride is used as a refrigerant and, in addition to the above symptoms, causes convulsions and oliguria.

Carbon tetra-chloride rarely causes narcosis when encountered in industry, but vomiting commonly occurs after exposure, and the symptoms of an "acute abdomen" are occasionally produced by direct action on the bowel causing spastic contraction. There is epigastric pain and vomiting of sudden onset, associated with increasing tenderness in the loins. The urine is scanty, and contains red blood cells. Carbon tetra-chloride poisoning is always

serious even though the initial symptoms are slight. The patient should be kept at rest and all measures for liver function failure (see page 82) used. Both **methyl bromide** (see also page 374) and **carbon tetra-chloride** are used in fire extinguishers. In addition to the symptoms of solvent poisoning described, methyl bromide burns the skin.

NITRITE EFFECT.—**Nitro-glycerine** is a well-known oily explosive liquid which forms methæmoglobin in the blood, and causes dilatation of the blood vessels, a fall in blood pressure, and slowing of respiration. Severe headache follows the inhalation of vapour, but acute poisoning is rare in industry. Amyl nitrite has a similar effect. The symptoms usually subside satisfactorily if the patient is kept lying down, and the effects may be alleviated by injection of strychnine 4 to 8 mg. (gr. $\frac{1}{16}$ to $\frac{1}{8}$). Artificial respiration may be necessary.

MISCELLANEOUS POISONS

Lead.

This is a widespread occupational hazard to workers employed in the extraction, smelting, refining, melting and use of metallic lead and its compounds. Lead is principally absorbed via the respiratory tract, following exposure to fumes and dust, but absorption also occurs from the gastro-intestinal tract, and from the skin in the case of tetra ethyl lead.

Lead *intoxication*, indicated by palsy, colic and encephalopathy, must be clearly distinguished from lead *absorption*. The urgent symptoms of colic and encephalopathy must be attributed to lead if a worker exposed to lead shows other evidence such as anæmia, punctate basophilia, a blue line on the gums, muscular tremors, paralysis and the presence of lead in the fæces and urine. In lead poisoning the urinary lead concentration is generally greater than 0.150 mg. per litre, the upper limit for normal persons being 0.08 mg. per litre.

LEAD COLIC.—This is characterised by sudden intense gripping pain, around or below the umbilicus. The patient is cold, pale and perspiring. The abdomen is tense, but no true rigidity is present on examination between the spasms. There is a history of constipation, and sometimes of a prodromal period in which there has been anorexia, nausea and intermittent pain in the lower abdomen.

The local application of moist heat, enemas and a belladonna and magnesia mixture are classical remedies. Symptomatic relief is promptly obtained by the intravenous or intramuscular injection of 15 ml. of a 20 per cent. solution of calcium gluconate.

Treatment with Calcium Disodium Versenate (the calcium chelate of disodium ethylene diamine tetra-acetate) should be started. Lead in the body displaces calcium from Calcium Disodium Versenate and is bound in a soluble non-toxic chelate and rapidly excreted in the urine. The symptoms of lead poisoning have been relieved and lead removed from the blood and tissues without toxic effects from the C.D.V. or from the mobilised lead.

The indications for the use of C.D.V. are acute and chronic lead poisoning (including lead encephalopathy) and the prevention of exacerbation of symptoms in chronic lead poisoning. Administration is by intravenous drip 5 ml. (0.2 G. per ml.) being diluted with 250-500 ml. of Injection of Sodium Chloride B.P. or Injection of Dextrose B.P. The concentration of the drug should not exceed 3 per cent. and the diluted solution should be administered in one hour, twice daily for five days. Treatment may be stopped for two days and repeated for another three days if considered necessary. The dosage is based on body weight.

Maximum daily dose should not exceed 1 G. per 30 lb. body weight.

Maximum dose per hour should not exceed 0.5 G. per 30 lb. body weight.

Maximum weekly dose should not exceed 5 G. per 30 lb. body weight.

The oral route may be used in chronic lead poisoning and is convenient for prophylaxis or treatment of mild exacerbations. The dose is 4 G. daily in divided amounts (0.5 G. tablets) for the average adult patient. Children should receive 1 G. per 35 lb. body weight daily.

LEAD ENCEPHALOPATHY.—This is now almost unknown in this country, but has been described following exposure to tetra ethyl lead and prolonged exposure to inorganic lead.

Tetra ethyl lead is absorbed through the intact skin, and readily through the pulmonary epithelium. Being highly lipoid-soluble, it accumulates in the nervous system, producing acute poisoning. The most important symptoms are restlessness, trembling, twitching, ataxia, convulsions, and coma. They result from

a widespread stimulant action on the central nervous system. Delirium and delusions may occur, particularly if the onset is abrupt. Constipation is not a feature of tetra ethyl lead poisoning, nor is stippling of the red cells usual. The pulse rate is slow, the temperature subnormal, and both the systolic and diastolic blood pressures low. The diagnosis is based on a history of exposure, and with the symptoms described, may be confirmed by the presence of lead in the faeces and urine.

In treatment it is important to cleanse the skin by washing with spirit, followed by soap and water. Other measures are symptomatic, being directed to supplying fluid and ensuring sleep. In severe cases it is necessary to maintain an adequate fluid intake by means of dextrose and saline intravenously. Delirium may be relieved by 4 to 6 fl. oz. (114-170 ml.) of a 50 per cent. solution of magnesium sulphate given as an enema (*see page 198*). Pentobarbitone B.P. (Nembutal) 0.1 to 0.2 G. (gr. 1½ to 3) may be given and repeated as required. Strict nursing supervision is necessary in view of the suicidal tendencies which are sometimes shown.

Metal fume fever.

This is a form of acute industrial poisoning which occurs most commonly in brass casters, and is known as "brass founders' ague." It also occurs in people exposed to finely particulate metals containing zinc, and this is the essential cause. The initial symptoms are dryness of the throat and irritation of the respiratory tract, with some cough. Headache, fever, and chills, pains in the limbs, and sweating occur later. The attack usually subsides in 4 to 6 hours, and the patient is restored to normal. Treatment is symptomatic and no specific measures are necessary.

Arseniuretted hydrogen (Arsine, AsH₃). (*see also page 378*)

This is produced in industries where arsenic-containing metals and acids react. It is one of the few industrial poisons which may cause sudden death, being quickly absorbed through the lungs. It causes rapid and severe hæmolysis. The early symptoms are faintness, weakness, and intense headache, with shivering and severe abdominal and muscular pains. An early sign is hæmoglobinuria, which is followed by jaundice, anæmia and suppression of urine.

Treatment consists in the administration of oxygen, alkalis and copious fluids. British anti-lewisite (BAL) (Dimercaprol) is

effective against arsenic (and also against mercury and cadmium). Its use should therefore be considered (*see page 265*). Blood transfusion is indicated where there is severe anæmia and certainly if the hæmoglobin is below 60 per cent., or the red cells below 4,000,000 per cu.mm. It is necessary to perform the transfusion slowly to avoid circulatory overloading (*see page 48*). Liquids and glucose should be given freely by the mouth or rectum. Sodium citrate and sodium bicarbonate should be given in sufficient amounts to maintain an alkaline urine; 2 G. (gr. 30) of each every hour until the urine is alkaline, and thereafter the same dose four-hourly. It is believed that alkalis prevent blockage of kidney tubules by acid hæmatin. Oxygen should be given if there is anæmia since anoxæmia may be present in the absence of cyanosis. In severe cases, death is often preceded by anuria. The patient should be watched until the blood and urine are normal. When damage to the liver, spleen, and kidneys occurs it is probably caused by arsenic itself, rather than the effects of anoxia and blockage of the kidney tubules.

Carbon bisulphide (CS_2) and hydrogen sulphide (H_2S).

The effects of these substances are similar, but hydrogen sulphide causes the more acute symptoms. Exposure may occur in artificial silk workers, tanners, sewer men and chemical workers. Carbon bisulphide is used as a solvent for rubber, and is more commonly a cause of chronic poisoning.

Exposure to high concentrations of hydrogen sulphide may cause sudden death, but it is well to remember that this occurs but rarely in industry; HCN and arsine are the two other substances having a similar effect.

Absorption is mainly via the lungs, but carbon bisulphide is also absorbed through the skin. The essential action of both substances is paralysis of the central nervous system. The symptoms of acute poisoning are intoxication and narcosis, sometimes preceded by headache, vertigo and general weakness. The characteristic smell may be little noticed even in a high concentration of the gas. Mental disturbances such as agitation and hallucinations are common in cases of exposure to H_2S and CS_2 . Even with serious symptoms, recovery is the rule, and treatment is along general lines, the essentials being the administration of oxygen, combined with artificial respiration where necessary. Being a highly diffusible gas with a low molecular weight H_2S is not readily

adsorbed on to activated carbon. The standard cannister respirators therefore offer no protection against the gas.

Phenol.

This is absorbed by the skin and lungs, and fatal poisoning has resulted. Headache, vertigo, nausea, tinnitus, faintness, excitement, convulsions, and respiratory paralysis may occur.

Treatment must be directed towards removing any skin contamination by washing with soap and water. Subsequently, oxygen and artificial respiration should be employed if indicated.

Insecticides.

Some of the many chemicals used in agriculture are potentially dangerous to man. Though the plant hormones, chlorinated hydrocarbons, petroleum, metallic and acid solutions may, if used wrongly, cause illness and burns of exposed areas, they do not commonly cause serious toxic symptoms. Arsenical compounds and lead arsenate rarely cause poisoning.

Nicotine is used in industry for the manufacture of insecticides. It is absorbed readily through the skin and mucous membranes and causes excitation and then paralysis of the central nervous system. The immediate symptoms are nausea, vomiting, sweating and dyspnoea. If a concentrated solution is spilled on the skin it must be removed immediately by scrubbing with soap and water. Artificial respiration may be necessary for a prolonged period.

Dinitro-ortho-cresol is an important compound used as a weed-killer, insecticide, ovicide and fungicide. Toxic symptoms and deaths have occurred during its manufacture and also when used in an oily solution during the winter for spraying fruit trees. In the spring and summer strong aqueous solutions are sprayed on cereal crops and cases of poisoning are particularly liable to occur in hot weather. Absorption occurs through the lungs, but also through the skin and eyes and alimentary tract. D.N.O.C. accelerates cellular metabolism similarly to dinitrophenol, formerly used as a slimming agent.

Early toxic symptoms are fatigue, insomnia, thirst, excessive sweating and loss of weight, which in the fatal cases recorded were followed in a day or two by heat stroke with pyrexia, dyspnoea, anxiety, restlessness and tachycardia. Death occurs rapidly from respiratory and cardiac failure. Toxic symptoms can

be prevented by strict observance of the instructions for handling, which include protective clothing, washing facilities, medical supervision and decontamination of machinery and equipment. Treatment of early symptoms is symptomatic, but D.N.O.C. is a cumulative poison which is excreted slowly and all slight or suspected cases should be removed from contact for at least six weeks. Yellow discolouration of the sclera is a sign of the absorption of potentially dangerous amounts. The patient should be at rest and given barbiturates. The excessive sweating and pyrexia are treated by oxygen, fluids and cold spraying. It is claimed that the intravenous administration of 10 ml. of a 2 per cent. solution of sodium methyl thiouracil reduces the increased metabolic rate.

Organic phosphorus insecticides (Tetra-ethyl-pyrophosphate [T.E.P.P.], Para-nitrophenyl-diethyl-thiophosphate [Parathion], Hexa-ethyl-tetraphosphate [H.E.T.P.], Bis-dimethylamino-phosphorus anhydride [Schradan, Pestox III, or O.M.P.A.]).

These compounds have an increasing use in pest control as sprays, dusts, or aerosols and may be absorbed into the body through the lungs, skin and alimentary tract. They all cause similar toxic effects. They are cholinergic by inhibiting cholinesterase and cause stimulation of the parasympathetic nervous system (muscarine effect) and of the motor nerves (nicotine effect), and have a direct action on the central nervous system.

Lowered values of blood and plasma cholinesterase occur after exposure to phosphorus insecticides. Most results indicate that levels below 50 per cent. of normal suggest intoxication and values of between 50 to 60 per cent. of normal are suspicious. There are several established methods for the estimation of serum cholinesterase. The principle is to incubate the patient's plasma with acetylcholine under standard conditions, when the acetylcholine is broken down with the production of acid. The hydrogen ion concentration is then measured with a pH meter (electrometric method) or by a colour change (colorimetric method). Liver disease, pernicious anæmia and malnutrition may also give a lowered plasma cholinesterase and it is raised in some cases of myasthenia gravis.

The early symptoms of poisoning are anorexia, nausea, constriction of the chest, giddiness, headache and disturbance of vision from constriction of pupils. Later symptoms are abdominal colic, pallor and sweating, muscular twitching, incontinence followed by restlessness, pulmonary œdema, coma and respiratory

paralysis. The muscarine-like symptoms, but not the nicotine effect and the action on the C.N.S., can be relieved by the intravenous or intramuscular injection of atropine sulphate 1 to 2 mg. (gr. $\frac{1}{64}$ to $\frac{1}{32}$) repeated at hourly intervals as necessary. Postural drainage, oxygen under pressure (*see page 574*) and artificial respiration (*see page 543*) may be required for the treatment of pulmonary oedema and respiratory paralysis. The skin and eyes, if contaminated, should be thoroughly irrigated with water. Prevention of poisoning calls for washing facilities, protective clothing and thorough instruction and supervision of spray workers.

HYDROFLUORIC ACID BURNS

Concentrated hydrofluoric acid causes skin lesions varying from erythema to severe burns. Droplets of anhydrous hydrofluoric acid cause penetrating burns in direct proportion to their size. Pain, which may be delayed, is severe. The burnt area is at first red and later blanched in the centre. If untreated it progresses to necrosis.

Treatment is by prompt flooding with large quantities of water for several minutes. Magnesium oxide paste (magnesium oxide 1 part mixed with medicinal paraffin or glycerine 2 parts) and a dressing should be applied. When central blanching occurs 10 per cent. calcium gluconate should be injected into, around and beneath the affected area. The injection must be thorough and made under anaesthesia if necessary. Relief of pain is usually immediate. Thereafter the lesion should be treated as a thermal burn (*see page 431*).

A. THELWALL JONES.

CHAPTER XX

Medical Emergencies at Sea

THE situation confronting the doctor when an urgent medical illness occurs on board ship has always been complicated by environmental factors. Of these, dirt and under-nourishment have largely been abolished, but rough weather, seasickness and shipwreck still threaten the sailor. In addition, scientific improvements have brought their own dangers, and to replace the scurvy, the fever and the fluxes of the sixteenth and seventeenth centuries come the fumes, the bends, the cramps and the blasts of modern times. There has been little fundamental change, however, in the nature of the mariner, and in the restricted world of ship life he thrives on rumour, loves gossip, and still retains some of the superstitions of the past. Unlike most forms of human society, the social structure in a ship has remained feudal, all owing allegiance to the master, and forming, as Dudley says, "a special herd in a special environment."

The importance of these points to the doctor is to show him how necessary it is that he should combine old wisdom with new knowledge. He must allay apprehensions and inspire confidence, as well as know the treatment of diseases met at sea. Discipline too is essential to the working of the ship, and he must therefore do nothing that would detract from the authority of the master and the ship's officers. He should take action in emergency, if necessary asking the advice of doctors travelling as passengers, and if circumstances warrant it he should let it be known that requisite action is being taken. On the other hand, elaborate treatment is unsuitable at sea, and should not be entertained.

MEDICAL AID TO OTHER SHIPS

Fairly often, medical advice is sought by a ship not carrying a doctor. If in convoy, the message may be received by visual signal or loud hailer, but normally, radio transmission is used, often allowing direct conversation and for which special codes have been arranged by the Ministry of Transport. The master of the ship seeking advice is instructed to acquaint the doctor with his medical resources and the type of medicine chest carried; he has

with him the *Ship Captain's Medical Guide* (19th edition, 1952, price 1 guinea, with Amendments 1 (1953) and 2 and 3 (1955)), and reference to this and the 1931 *International Code of Signals* (Medical Section, Vol. 11, pages 233 to 246) will show how details of a case should be given to the doctor. Whenever possible, the doctor should reply with the diagnosis, directing the master to the treatment in his book; he should make sure that any drug ordered is actually carried on board. The doctor may also have to give an opinion as to whether the ship should continue on her course or make for the nearest port. Since he cannot examine the patient, instructions given by wireless do not involve him in any legal responsibility either through errors in transmission or from any other cause.

When advice is asked about a fever, Paludrine, mepacrine or quinine should be ordered if there is the slightest possibility of malaria. Since men collapsing in an engine-room in the tropics are very often suffering from cerebral malaria and not heat stroke it is necessary in such cases to give instructions on how to inject quinine or mepacrine.

When ships are close together it may be possible for the doctor to visit the patient by ship's boat. Failing this, various devices such as a Coston gun or a buoy have been used for sending drugs.

SEASICKNESS

In spite of innumerable treatments, this malady persists, and it is fair to say that, in very rough weather, most passengers in small ships will still be sick. Sufferers react in different ways; some like to go on deck, while others will not leave their cabins; some take glucose, others champagne, while others put their trust in tight abdominal binders.

While no treatment is specific, hyoscine is well worth a trial; one hour before rough weather is expected 0.65 mg. (gr. $\frac{1}{100}$) should be taken by mouth; this may be followed at six-hourly intervals by 0.32 mg. (gr. $\frac{1}{200}$) for 48 hours. It produces in many people a remarkable feeling of stability, and seasickness can often be avoided until "sea legs" are gained. Side effects are dryness of the mouth and drowsiness, and sometimes interference with accommodation for near vision. Avomine (May & Baker) 25 mg. by mouth before sailing has given good results.

In severe cases it is wise to be on the lookout for evidence of dehydration, and to anticipate it by ordering frequent sips of glucose drinks. Very occasionally intravenous dextrose and saline may be necessary, but however extreme the prostration, death practically never occurs in uncomplicated cases.

When symptoms are severe, other conditions may also be present. Thus, when meningitis occurs at sea, an all too common story is that "the doctor first thought it was seasickness." The same is true of diphtheria, when the sore throat has been attributed to persistent retching, with tragic consequences. Less important, but also worth remembering, is that the vomiting of pregnancy and seasickness may occur together.

SHIPWRECK

Even in peace-time, problems resulting from shipwreck form some of the most important medical emergencies, and the ship's surgeon may be called upon to show the qualities of a seafaring man.

Precautions against shipwreck are the responsibility of the master, but on ship's rounds a tactful enquiry about the water and food capacity of lifeboats may stimulate interest towards improvements. Thus, although on an average each boat carries 40 gallons (145 litres) of water, some masters have managed to improvise stowage for as much as 120 gallons (435 litres) per boat. Further knowledge about actual conditions in lifeboats (location of medical supplies, etc.) can be gained by the doctor taking part in exercises when a boat is lowered for a practice pull or sail. He should also make it his personal responsibility to see that a copy of the *Medical Research Council War Memorandum No. 8*, which deals with the preservation of life at sea after shipwreck, is stowed in each boat. He himself should be thoroughly conversant with the contents of the publication.

Before abandoning ship, everyone should try to take a long drink of water, and collect extra warm clothes. These are essential, even in the tropics, where it can be bitterly cold at night. Wet clothing, particularly socks and boots, should be dried as soon as circumstances permit. Theoretically, resuscitating the drowned will be the earliest problem but in practice it may be more important to prevent men who are struggling in the water from upsetting a fully-loaded boat. If the water is

very cold, men should be advised to swim or struggle as hard as they can for as long as they can. If they try to preserve their strength by clinging to wreckage, or floating on their life belts they will die more quickly from cold.

As water is the most important single factor in determining survival, attention must at once be given to rationing supplies. Each boat is scheduled to carry at least $5\frac{1}{2}$ pints, say 3 litres, per man. If there is likelihood of being in the boat for, say, a week, the ration recommended is 18 fl. oz. (500 ml.) per man daily. None should be given in the first 24 hours, then 6 fl. oz. (168 ml.) thrice daily until one pint per man remains, when the ration should be reduced to 2 fl. oz. (56 ml.) per man per day. Alcohol should be reserved for the wounded and moribund; for others it will only increase thirst and heat loss. Regular sleep should be obtained during watches off, but it may be necessary in periods of emergency for some, or all, of the boat's company to stay awake. At these times the use of 5 mg. tablets of dexamphetamine is recommended. One or two tablets of each, thrice daily, may be used.

The crossing of the Atlantic by Dr. Alain Bombard in 1952 on a rubber raft and his heterodox views on the drinking of sea water have been widely discussed. The consensus of opinion is that, although he has shown that man can exist for a long time at sea by courage and determination yet he has not produced acceptable evidence to support the drinking of sea water. The reasons for this are as follows:—

"A man deprived of an adequate intake of water continues to lose water through his skin, lungs and kidneys. As a consequence the osmotic pressure of his body fluid rises, and this, rather than the change in their volume, eventually causes death. Ingestion of salt, or of fluids such as sea water which contain salt in a concentration higher than is found in human urine, increases the hypertonicity and so hastens death. This is so whether the salt is retained in the body or excreted with expenditure of body water. Sea water also causes water to move from the cells to the extracellular compartment. This probably explains the apparent benefit which has been reported after sea water drinking for periods of up to four days, since most of the signs of dehydration actually depend on the state of the extracellular compartment only. Intracellular dehydration is, however, the cause of death and of the signs of failure of the central nervous system which apparently precede death." Hervey (*Brit. med. J.* 1954, 1, 1494).

Macdonald (*Brit. med. J.* 1954, 1, 1035) concludes: "Those who are cast away at sea who do not drink sea water and wait for the rain that saved Bombard will be more certain to survive than those who do drink sea water provided they are equally courageous."

Treatment after rescue.—Above all else, survivors need drink, food, warmth and prolonged sleep. For the last, phenobarbitone should be given for several days, but it should be remembered that after exposure to extreme cold susceptibility to sedatives is

increased, and smaller doses than usual are effective. A big meal should not be taken for three or four days; bread and milk, or thick soup is recommended to start with. If dehydration is severe, a slow dextrose saline infusion is better than either blood or plasma, both of which increase viscosity.

Survivors are prone to curious hallucinations, possibly caused by cerebral anoxia. If troublesome, the effect of oxygen should be tried. Certain other conditions often, but not exclusively, found in survivors and caused by exposure, are as follows:—



FIG. 36

Immersion foot.

(*Surgery of Modern Warfare.*)

(1) **Immersion foot.**—This is particularly liable to occur in men who have stood on a partially submerged raft for several days. The feet become swollen, numb and white, and in severe cases ulceration of the skin occurs, usually associated with salt-water boils (Fig. 36). The muscles are weak, and there is anæsthesia of the stocking or slipper type. Pain is marked only during recovery, when agonising burning sensations are experienced in the soles of the feet.

Treatment.—Little can be done in the way of prevention, though if men wear sea-boots and keep moving they are less liable to suffer.

When the condition has developed, the essential treatment is to keep the patient warm and his feet cold, as it is harmful to heat the affected part until its oxygen supply is adequate. Therefore, remove the patient's clothing and wrap him in warm blankets; do not allow him to walk. The feet and legs should be gently raised on pillows and left exposed to the air. Use an ordinary fan intermittently to play cool air on the feet, reducing the temperature to between 70° and 80°F. A bath thermometer wrapped in cotton wool can be used for controlling the treatment. After several days the pain and swelling are lessened and the air temperature can be raised and gentle exercises ordered. Abrasions should be dusted with sulphanilamide powder and protected by light dressings. It is wise to give tetanus anti-toxin. Diet should be of a high calorie value and it is probably helpful to give supplementary vitamins. (*See Ministry of War Transport Notice No. M.226 for details of treatment of immersion foot*).

(2) **Frostbite.** (*see page 488*)

(3) **Snow-blindness.** (*see page 410*)

(4) **Sunburn.**—Severe sunburn causes blistering of the skin and when very extensive there is also constitutional disturbance—fever, malaise and headache—(popularly known as “sun-stroke”). Many cases occur on pleasure cruises despite all warnings. Those with fair skins suffer the most.

In the Services, reporting sick was prevented by making the condition a punishable offence. Obviously this is not practicable in civilian life but the habit of sleeping in the sun for long periods should be discouraged and instructions circulated giving safe lengths of time for sunbathing. Red or brown grease paint affords some protection. When insects and sun combine to attack the skin the following lotion is useful in preventing bites:—

R	Phenol	0.4 ml. (m 7)
	Thymol	0.32 G. (gr. 5)
	Weak Solution of Iodine	...			3.5 ml. (m 60)
	Camphor Water		to 28 ml. (1 fl.oz.)

For established sunburn, blisters may be opened under aseptic conditions and the raw areas covered with calamine lotion containing 1 per cent. phenol or 2½ per cent. of solution of coal tar. This will allay itching but sedatives such as phenobarbi-

tone 32 to 65 mg. (gr. $\frac{1}{2}$ to 1) may also be necessary to ensure sleep.

THE EFFECTS OF EXCESSIVE HEAT

It is a common event in any ship for a man to collapse at his work, or for a passenger to complain of a "turn," "dizzy spell" or "black-out." When this happens in hot weather, the question of how far the condition may be attributable to heat must be considered. The doctor, therefore, should know the symptoms and signs of heat affections—not only in order to treat these conditions but also to avoid the repercussions of unfortunate actions such as stripping and plunging into iced water influential passengers who are only suffering from the "vapours" in their convalescence from some acute debilitating illness.

The main syndromes are described under "Emergencies in Tropical Medicine" (page 338).

THE MANAGEMENT OF EPIDEMICS

It is impossible to lay down hard and fast rules for handling infectious disease because circumstances vary with the number and type of cases. Passengers can be isolated in spare cabins as far aft as possible, while for the ship's crew a hospital is usually provided. In smaller vessels, improvised isolation can be arranged by rigging a tent over the after hatch, constructional details being given in the *Ship Captain's Medical Guide*.

In severe cases it will be necessary to detail one or more members of the crew to act as sick nurses. These men must observe the usual precautions, such as wearing special overalls or oilskins while at work, and they should come into contact with the rest of the crew as little as possible.

Smallpox. (see also pages 316 and 611)

The nurse should either have had the disease or have been recently vaccinated successfully. Everyone on board should be vaccinated or re-vaccinated. Only those who can show documentary evidence of successful vaccination within the preceding 12 months should be excluded, and the presence of a scar should be checked. Protection of contacts put ashore at intermediate ports before the discovery of smallpox should not be overlooked. In practice, difficulties may arise, because enough lymph may

not be available or because it has lost its potency (*see page 611*). On arrival in harbour, the Port Medical Officer will inspect the vessel, and give instructions and help concerning vaccination, the disposal of the sick, and the follow-up of contacts. Many ships carry special outfits for sending material for the diagnosis of smallpox (obtainable from Messrs. R. B. Turner, 9 Eagle Street, London, S.W.1).

The method of collection of material is as follows.

Maculo-papular rash.—Clean skin with ether or methylated spirit. Scrape at least six lesions with needle and make six smears on four glass slides. Allow smear to dry in air. *Do not heat*. Place each pair of slides face to face but separated so that they will not stick together, mark side of slide on which smear is made and return to box. Return needle in tube.

Vesicular and pustular stage.—Remove tops of 6 lesions and place in small bottle. Collect fluid in capillary tubes and replace in large bottle. Scrape base of lesions 4, 5 and 6 and make smears as above.

Crusting stage.—Remove as many scabs as possible up to 12 and place in small bottle.

The container must be sent by air mail with Express label to Virus Laboratory, Central Public Health Laboratory, Colindale Avenue, London, N.W.9.

(*See Ministry of War Transport Notice No. M.231* for details of diagnosis and precautions to be taken in smallpox).

Bacillary dysentery.

This is one of the commonest infections occurring on board ship, and on vessels bound for the East it often flares up among the crew and spreads to the passengers within a few days of leaving Suez. As in practice ashore these minor epidemics are usually transient, and the carrier of the infection rarely identified. Enquiry should be made about recent diarrhoea among the food-handlers of the crew, and the standard of personal cleanliness of the kitchen staff must be closely scrutinised. Members of the crew who have been ashore at Mediterranean ports, and taken food there, should be considered suspect. Frequently, on these occasions passengers come forward with suggestions for controlling the epidemic; for obvious reasons they should be given a careful and courteous hearing. Drinking water, bilges, rats, flies and bugs are invariably indicted and useful information may sometimes be

gathered. Extra cleaning and scouring should be ordered and stringent measures adopted to eliminate flies and vermin. It is usually worth while to publish a statement indicating that suitable steps have been taken, thus reassuring passengers who may otherwise be apprehensive and lose confidence in the medical arrangements on board and in the efficiency of the crew as a whole. (*For treatment of bacillary dysentery and other types of acute diarrhœa, see pages 71 and 316*).

GENERAL PRECAUTIONS.

(i) Ships calling at malarious ports should be as far off shore as possible, as mosquitoes may be carried long distances from the shore by wind.

(ii) Communication between the ship and the shore should be cut down to an absolute minimum, or even forbidden, between the hours of dusk and dawn. This applies especially to loading barges, fruit boats, etc., which are likely to carry infected mosquitoes from the shore to the ship.

(iii) If possible, portholes, doors, ventilators, etc., should be mosquito-proofed. This measure should be completed a day or so before arriving in a malarious area, and kept in operation for at least four days after leaving. Failing this, mosquito nets should be supplied to each member of the crew and to every passenger.

(iv) No one should be allowed ashore except for very urgent reasons, and no one should be allowed to remain ashore after dusk.

(v) No one should be allowed to sleep on deck, unless provided with a suitable mosquito net, which must be used in a proper fashion to prevent mosquito bites.

(vi) All lights not absolutely essential for the working of the ship should be screened, as these are liable to attract mosquitoes from long distances.

(vii) After sunset all persons should wear sufficient clothing to protect the whole body from mosquito bites (long sleeves, long trousers, two pairs of socks, etc.). Fans are useful in helping to keep mosquitoes away, but are not a certain protection. The exposed portions of the body (wrists, behind the ears, ankles, back of neck, etc.) should be smeared with culcifuges after dusk. The best is dimethyl phthalate (DMP). It must be remembered, however, that the effects of these substances only last for about three hours or so, and that the application must then be renewed.

(viii) Cabins, bathrooms, etc., should be sprayed before bedtime and in the early morning. Special attention should be given to all dark corners, the inside of wardrobes, spaces under bunks, behind baths, etc. Insecticides containing DDT (Dicophane) or "Gammexane" and pyrethrum are especially useful. Sticks of incense burning in cabins will also tend to keep mosquitoes away.

(ix) Curtains and other materials hanging in the cabins should be well shaken and any mosquitoes destroyed before turning in.

(x) Bathing should be carried out during the hours of daylight and not after dark.

(xi) All persons feeling "off colour" should report to the Medical Officer at once, as this may be a premonitory symptom of a malarial attack, and early treatment is absolutely essential.

MEDICAL PRECAUTIONS.—Prophylactic drugs should be taken if the chances of infection are great. A responsible officer should see that all persons *receive and swallow* the prescribed dose. Proguanil B.P. (Paludrine) should be given to adults and children over 10 in doses of 1 tablet (0.1 G.) twice weekly. This will suppress benign tertian malaria and will give complete prophylaxis against malignant tertian malaria. If Paludrine is not available, mepacrine should be given in doses of one tablet (0.1 G.) daily after food and followed by a copious draught of water. Suppressive treatment should be started ten days before the malarious port is reached and continued for thirty days after the risk of infection is passed. Smaller doses are necessary for children under 10,

according to the usual formula— $\frac{\text{Age}}{20} \times \text{adult dose}$, but no child need be given less than 0.025 G. (i.e., $\frac{1}{4}$ of a 0.1 G. tablet) of proguanil (Paludrine).

Specific treatment.—Depending on the drugs available any of the following treatments may be used.

1. Paludrine 0.1 G. three times a day for 10 days or 0.1 G. twice a day for 14 days (malignant tertian malaria). A single dose of 0.3 G. will end an attack of benign tertian malaria but suppressive therapy should be given.

2. Quinine gr. 10, three times a day for seven days.

Quinine Sulphate (or Hydro-

chloride) ... 0.65 G. (gr. 10)

Citric Acid ... 2.0 G. (gr. 30)

Magnesium Sulphate ... 1.3 to 4.0 G. (gr. 20 to 60)

Spirit of Aniseed ... 0.6 ml. (m 10)

Simple Syrup ... 15 ml. ($\frac{1}{2}$ fl. oz.)

Water ... to 30 ml. (1 fl. oz.)

3. Mepacrine 0.1 G. three times a day after food for five to seven days.

4. Quinine as in 2, for three days, and then mepacrine as in 2, for five to seven days.

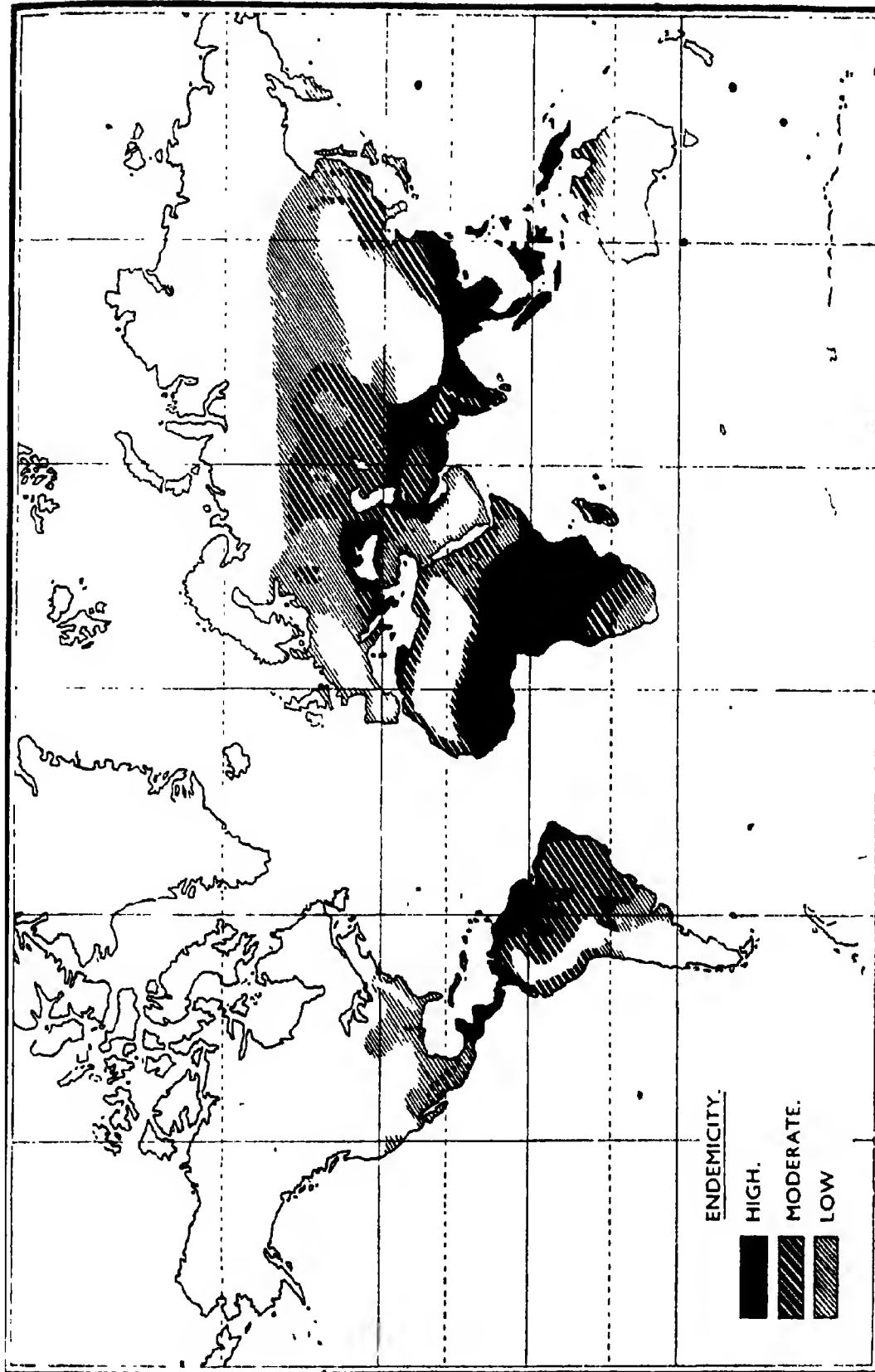


FIG. 37 —Showing where malaria may be expected.

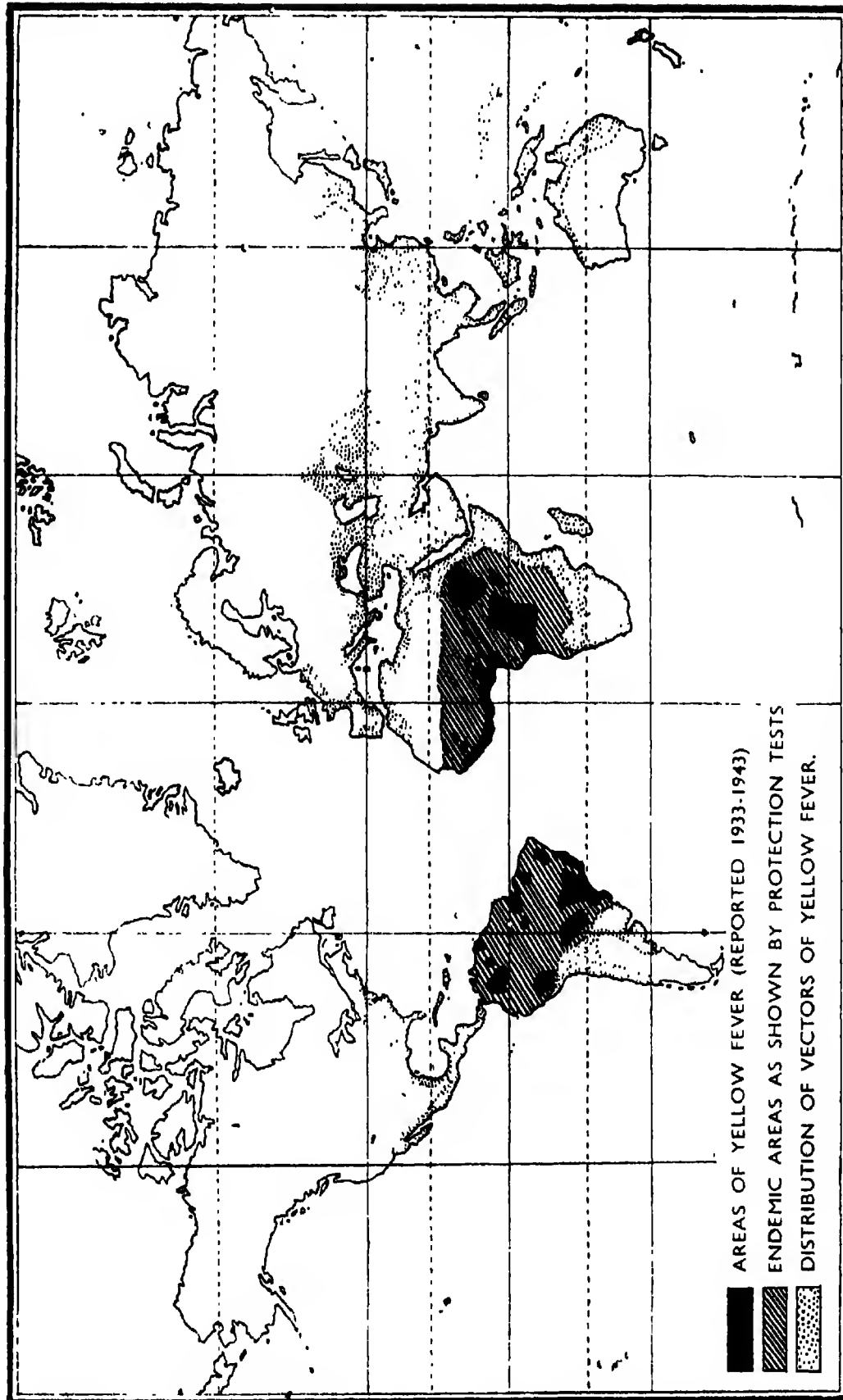


FIG. 38—Showing where yellow fever occurs. The, light stippled areas indicate “yellow fever receptive areas,” i.e., where the disease does not exist but where conditions would permit its development if introduced.

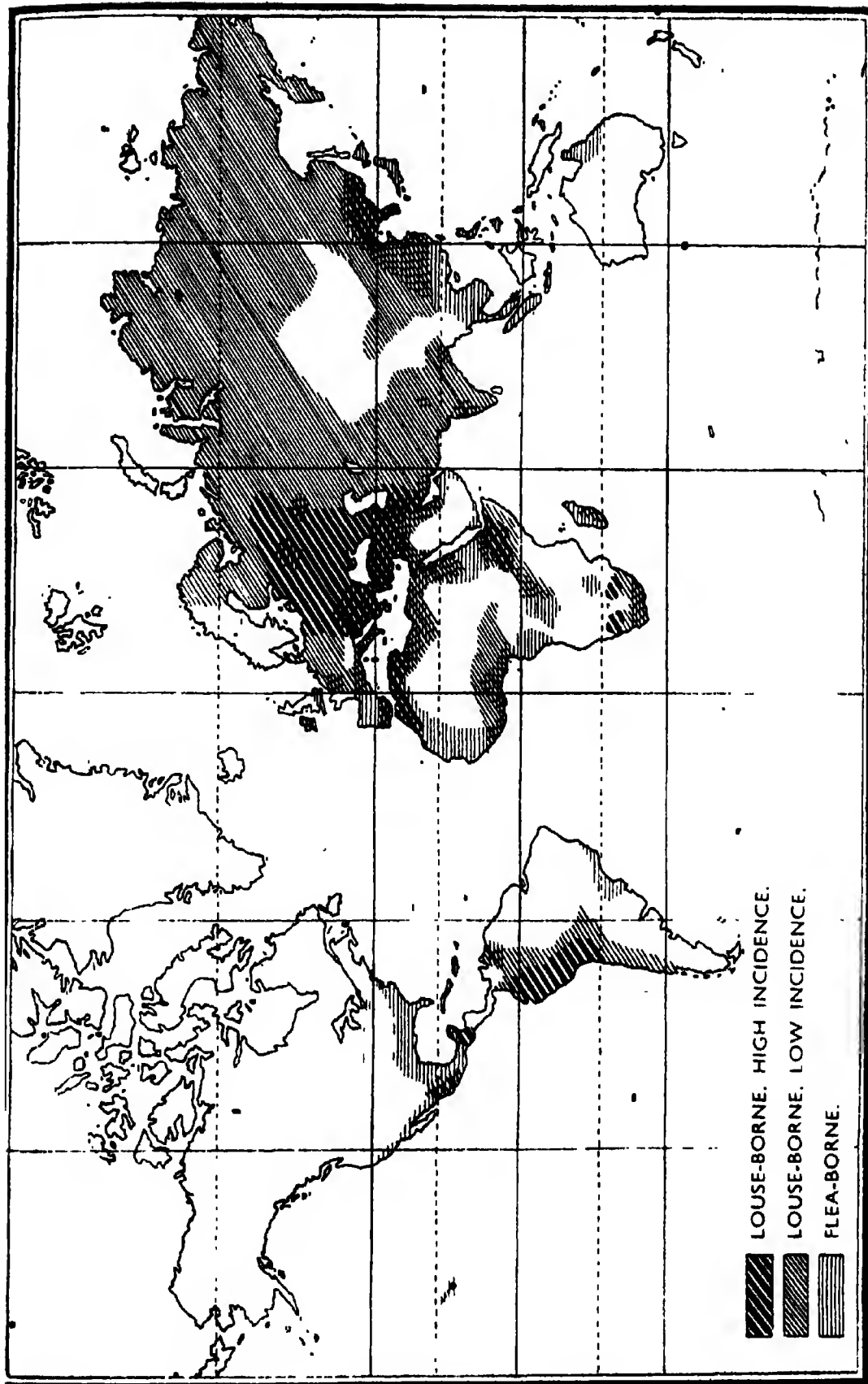


Fig. 39—Showing where typhus fever may be expected.

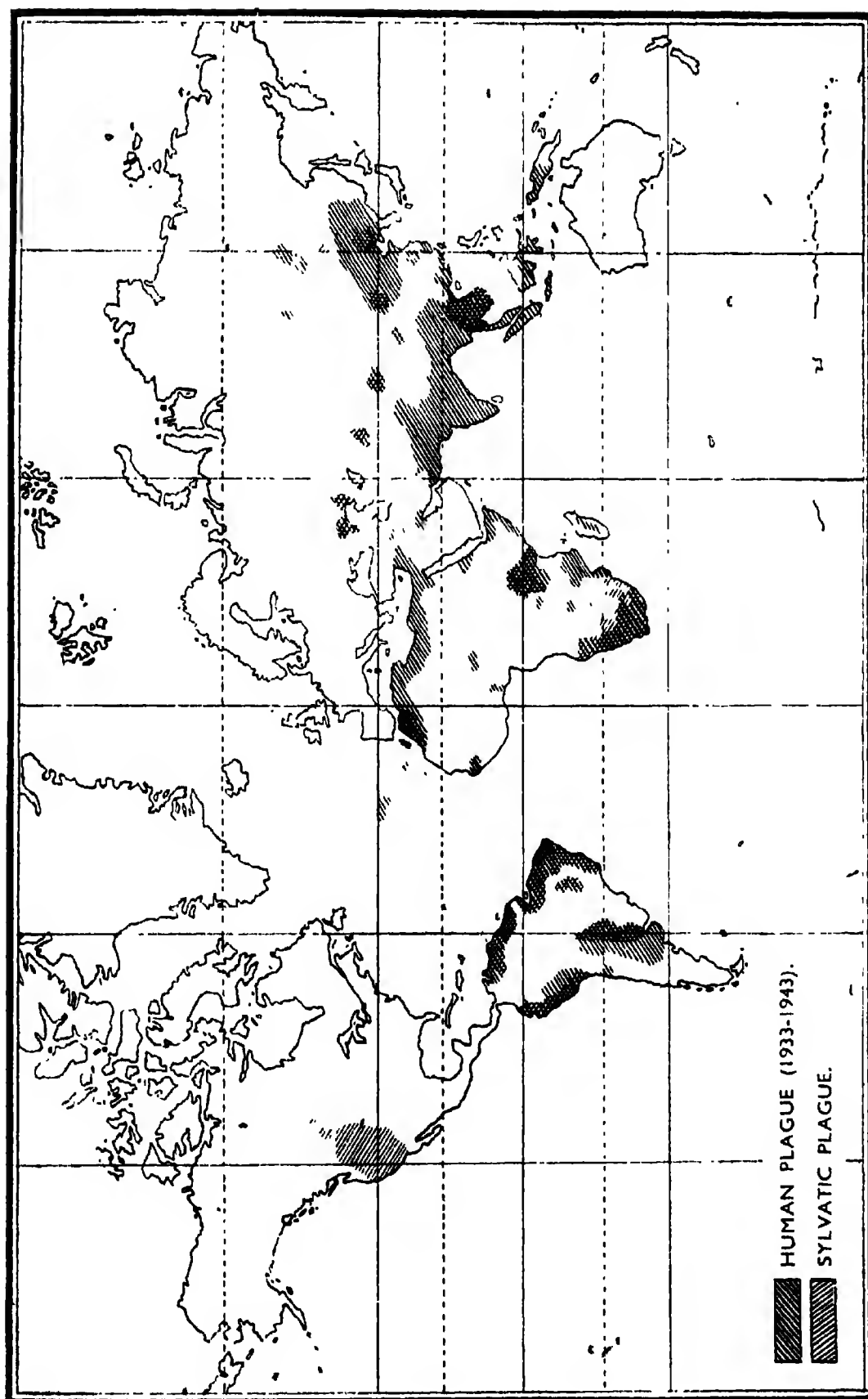


FIG. 40—Showing where the plague may be expected.

Treatment of cerebral and other forms of pernicious malaria is described on page 326 *et seq.*

The four maps show in which part of the world some of the commoner tropical diseases can be expected to occur (Figs. 37, 38, 39 and 40).

A NOTE ON WATER AND FOOD SUPPLIES.—Sometimes the master may be obliged to take in water, the source of which is suspect. It should then be chlorinated by adding one part of chloride of lime to 250,000 parts of water, *i.e.*, one teaspoonful to 200 gallons. For successful chlorination 0.2 to 0.5 parts of free chlorine per million parts of water must be present. The concentration can be determined with the Chlorotex reagent (British Drug Houses) by mixing 50 ml. of water with 5 ml. of reagent in a cylinder and comparing the colour after one minute with that of a chart. Pink indicates 0.2 parts and red 0.5 parts per million. It may be necessary also to recommend chlorination in a typhoid or dysentery epidemic. Any objectionable taste may be removed by adding potassium permanganate (0.2 to 0.8 parts per million) before, with, or after treatment. To disinfect small quantities of water add 2 or 3 drops of Weak Solution of Iodine B.P. to a quart. Stir thoroughly and allow to stand for at least 30 minutes.

The criteria of good and bad food are given in the *Ship Captain's Medical Guide*. A point, however, that needs to be stressed is the danger of buying fresh fruit and vegetables in foreign ports. They are very likely to carry dysenteric infections which are always endemic in the East during the warmer months. An order forbidding trade with fruit boats cannot be enforced, and it is probably better simply to insist that the food-stuffs should be immersed in weak potassium permanganate solution, and then washed in running water if they are not to be cooked.

Stench.

A compartment which has become foul-smelling should be washed out with chloride of lime ("bleach") and water (1 in 20). Metals must first be covered with grease to prevent corrosion. In war-time, corpses may have to be dealt with. Damp sawdust and bleach powder, 5 to 1, will render the atmosphere tolerable. A similar situation might arise with decaying carcasses in peace-time, and the wearing of respirators affords protection while the cleaning is being effected.

POISONING ON BOARD SHIP

A variety of poisons, more or less peculiar to ship life, may cause emergencies at sea.

Alcohol.

Acute alcoholism is common, especially when a ship is in port, and sailors may achieve an astonishing degree of coma, *not infrequently associated with head injury*. The condition is usually caused by the rapid consumption of an excessive amount of ordinary liquor, but where "doping" is alleged, methyl

alcohol poisoning may have to be considered (*see page 12*). This should be suspected when dimness of vision and general symptoms come on after a latent interval of from 9 to 36 hours.

The treatment of a severe case of alcoholism in coma should be by stomach washout (*page 537*), using warm sodium bicarbonate, one teaspoonful to the pint; where the patient is conscious, vomiting should be encouraged and apomorphine hydrochloride subcutaneously 6.5 mg. (gr. $\frac{1}{10}$) is a convenient emetic.

Chronic alcoholism engenders difficulties of a different order. The doctor should keep aloof from alcoholics among the passengers, for although their company may be amusing, the association will not enhance his reputation.

Ethylene glycol.

This substance, used as a solvent for nitro-cellulose and "dopes," has been taken as an intoxicant and caused many deaths. It produces a hepato-renal syndrome of which anuria is a special symptom. Treatment is on the lines suggested on *page 82*.

Carbon monoxide poisoning on board ship may occur alone or in conjunction with intoxication by nitrous fumes (*see page 7*).

Carbon dioxide may be sufficiently concentrated in ships' holds to cause asphyxia (*see page 345*).

Nitrous fumes result from incomplete combustion of cordite and are therefore likely to appear when a mine or torpedo strikes a ship carrying ammunition. Symptoms and treatment are as described on *page 349*.

Carbon tetra-chloride may cause poisoning on board ship when used in firefighting, and minor symptoms have occurred when it has been used for cleaning clothes. They are described on *page 351*.

Methyl bromide is a special hazard in motor torpedo boats. Its heavy vapour is most effective against fires involving high octane spirit. In high concentrations, as in fire-fighting, pure gassing may occur, but in the recent war nearly all cases resulted from accidental inhalation of much lower concentrations of the vapour. For instance, slight leakage into the wardroom of a motor torpedo boat produced symptoms which were first interpreted as food poisoning. A few hours later two of those affected went into convulsions and died, while a third had staggering gait and other symptoms suggestive of alcoholic intoxication. The illness remained obscure until somebody thought of the fire extinguishers, and weighing of the cylinders showed that one was empty.

There is no effective treatment but continuous oxygen, and, where fits are occurring sedation with Thiopentone B.P. (Pentothal) or paraldehyde (*see page 597*) can be tried. In patients who recover, convalescence is often complicated by depression.

Methyl chloride, which is used in many ships' refrigerators, produces similar symptoms. Consciousness is quickly lost. Recovery sometimes follows artificial respiration. (*See Ministry of War Transport Notice No. 251 for details of treatment of casualties caused by refrigerant gases*).

Incidents of this kind emphasise the importance of thorough investigation whenever several men go sick with symptoms that are at all unusual.

UNDER-WATER HAZARDS •

(1) **Caisson disease.**—A caisson is a device for enabling a number of men to work on the sea bottom or alongside wrecks, usually at a stretch of from six to eight hours, at a depth of 60 feet or less, without the encumbrance of diving suits. Essentially it is a metal column, at the bottom of which men work in a special chamber. Water is prevented from occupying the working chamber by the counter-pressure of compressed air. Communication between the working chamber and the external atmosphere is effected by a series of airlocks, in which men are gradually compressed or decompressed.

Caisson disease results when decompression is carried out too quickly. Bubbles of nitrogen are formed, particularly in those tissues with a poor blood supply (joints), or those containing much fat (spinal cord and subcutaneous tissues).

A similar condition occurs, but more rarely, in divers who work at greater depths, but for shorter periods. The condition appears only when the pressure of the air is more than 18 lb. to the square inch, *i.e.*, below 40 feet of water.

SYMPTOMS.—

- (a) " Bends "—liberation of bubbles into the ligaments of joints causing pain and flexion. These may not come on for five to six hours after reaching the surface.
- (b) Patchy discolouration of the skin.
- (c) Abdominal pain, distension and vomiting.
- (d) Nervous symptoms—headache, vertigo, paraplegia, coma and convulsions.
- (e) Deafness caused by low pressure in the middle ear.

X-ray examination of men who have worked in compressed air will sometimes show infarcts in the shafts of the long bones (Fig. 41). These findings, though not entirely specific for Caisson disease, might afford supporting evidence in medico-legal cases.

Treatment.—Caisson disease can be prevented by gradual decompression according to the Admiralty instructions. For divers, a Davis Submerged Decompression Chamber containing oxygen is now often used. This the diver enters 60 feet below the surface (not deeper, to avoid oxygen illness), and he can then be quickly raised to the parent vessel with much less risk of decompression sickness.

When symptoms are present it is necessary to re-compress the patient to the original pressure at which he was working, and then to decompress slowly over a period of about five hours.



FIG. 41

Bone infarcts in a caisson worker. Age 52. Cramps in thighs, forearms, hands and abdominal wall 1 year. Was a tunneller for 18 years. Worked under pressures of 5 to 44 lb.

Recompression chambers are obtainable from Siebe Gorman & Co., Ltd., Davis Road, Kingston-by-Pass, Surbiton, Surrey, Telephone Elmridge 5900, who will also give the location of the nearest one.

If none is available, the emergency is best dealt with by placing the patient in a diving suit and lowering him to the bottom in the care of another diver.

Oxygen is also useful in "bends," and alkalis should be given as they increase the CO_2 combining power of the blood. In port it should be remembered that there is usually a practitioner available who has had practical experience in dealing with compressed air sickness and his advice should be sought.*

There seems to be no means of picking out men unduly susceptible to compressed air sickness other than by recording the effects of actual work on the job under pressure, as individual susceptibility varies from day to day and week to week.

(2) **Air embolism.**—This can complicate submarine escapes and should be dealt with as described on *page 26*.

(3) **Oxygen disease.**—Breathing of pure oxygen at a depth of 120 feet (four atmospheres pressure) produces acute oxygen poisoning because it interferes with the carriage of CO_2 from the tissues owing to lack of reduced hæmoglobin. Divers, and submarine crews breathing pure oxygen while awaiting escape by the Davis Submerged Escape Apparatus, are liable to be affected. Early symptoms are nausea and vertigo, twitching and loss of control of the lips, and tingling of the extremities. Within an hour, convulsions, coma and death follow. Relief is obtained by breathing ordinary air, but in the case of the submarine crew awaiting escape this introduces a risk of Caisson disease. Men brought to the surface usually* recover rapidly, convulsions ceasing and the patient falling into a deep stertorous sleep. Later there may be headache, vomiting and loss of memory, but no permanent injury.

Too rapid ascent of a diver breathing oxygen or a man in a Davis Submerged Escape Apparatus carries with it the risk of "burst lung" due to trauma of the alveolar walls by the oxygen rushing out of solution. Hæmoptysis, not necessarily fatal, occurs.

Note.

(a) "Burst lung" is not Caisson disease, since the subject is breathing pure oxygen. Workers in caissons and divers breathing *ordinary air* are *not* liable to acute oxygen sickness, since the partial pressure of oxygen necessary to produce symptoms would only obtain at a depth of 600 feet or more, which is outside the range of both types of worker.

(b) Chronic oxygen sickness (a pneumonia-like condition produced by breathing oxygen at a partial pressure of one atmosphere for many

hours) could theoretically affect divers breathing compressed air at a depth of 150 feet. Under ordinary working conditions this does not happen since exposure is not long enough.

(3) **Arsine and stibine poisoning.**

In the manufacture of submarine batteries, scrupulous care is taken to exclude arsenic from the lead plates, but during the last war there was at least one boatful of cases of poisoning. During charging, batteries evolve hydrogen which contains traces of arsine if the lead or sulphuric acid contain arsenic. If the boat dives at the end of a rapid charge the batteries continue to evolve gas and the crew may be poisoned.

A similar accident may result from the formation of stibine from antimony present in the grid of the battery. Both arsine and stibine are essentially odourless and their toxicities are similar. Their presence can be recognised by the fact that silver nitrate test papers turn black.

Symptoms and treatment are essentially the same as in similar poisoning in industry (*page 354*).

(4) **Immersion blast.**

Under-water explosions produced this additional hazard to survivors from ships in war-time. Ear drums may be ruptured and multiple hæmorrhages in the lungs cause dyspnœa and hæmoptysis. Perforation of, or hæmorrhages into the gut may occur in several places, either at the time of the injury or from 6 to 10 days later; in the delayed cases an infected hæmatoma is present.

Treatment. -Instruct sailors to swim on their backs as horizontally as possible if they are near a sinking ship when depth charges may explode. This will remove the air-containing organs as far as possible from the explosive wave which becomes intensified at any air-water boundary.

For the lung lesions absolute rest, morphine and oxygen are indicated. Where perforation is obvious, laparotomy should be performed, but nice judgment is required when the signs are less definite. If laparotomy is undertaken an inhalation anæsthetic should not be used owing to the serious hazards associated with blast lung.

MISCELLANEOUS CONDITIONS

The "acute abdomen" at sea.

Should a perforated peptic ulcer be diagnosed and a surgeon be judged to be unsuitable for gastric aspiration should be

After an injection of morphine the patient sucks a lozenge of amethocaine (65 mg. (gr. 1)) and the stomach is then emptied by a large stomach tube and a Senoran's evacuator (*page 541, Fig. 89*) or syringe. This tube is then withdrawn and a smaller one introduced and aspiration continued at half-hourly intervals for at least 48 hours. Parenteral chemotherapy should be started and dextrose saline given rectally, subcutaneously or intravenously. Sips of water may be taken but should be aspirated again at once. This treatment is more likely to be successful if adopted early, and if leakage of gastric contents has been minimal.

Toothache and gum infections.

Ships do not usually carry dentists, and the sea-going doctor will find it very useful to have some elementary knowledge about toothache, of which there are three main causes:—

(1) **Pulp infection.**—This occurs secondarily to a cavity, and the tissues surrounding the tooth are normal. Gentle probing will usually elicit which tooth is affected.

Treatment.—Unless the infection is gross, an attempt should be made to fill the cavity. Clean it out with an excavator after an injection of 1·2 to 2·3 ml. (20 to 40 minims) of 2 per cent. procaine round the tooth. Swab the cavity with oil of cloves and fill it with zinc oxide mixed with two or three drops of oil of cloves. This will relieve pain temporarily.

(2) **Periodontitis.**—This occurs secondarily to pulpitis. The tooth is tender on pressure, raised in its socket, and pus may appear round it.

Treatment.—Give very hot mouth washes of Solution of Chloroxylenol B.P. (Dettol), 12 drops in a tumbler of water; the liquid should be held by the tongue around the tooth. Never apply hot fomentations to the cheek lest the abscess be caused to point externally. Extraction is often necessary when pus is present, and should always be done under general anæsthetic, injections into infected tissues being dangerous.

(3) **Infection round erupting lower wisdom teeth.**—The patient complains of a dull pain, sometimes associated with trismus; there may be an inflamed gum flap over the tooth. Mouth-washes should be given and the flap painted with Weak Solution of Iodine B.P.

Ulcerative gingivitis (Vincent's angina).

This is characterised by sore and bleeding gums, and sometimes tonsillitis is present also.

Treatment.—In the acute phase stop brushing the teeth and discard the old toothbrush. Clean the teeth with cotton wool and wash the mouth out with Dettol or hydrogen peroxide thrice daily. Apply 10 per cent. chromic acid carefully to the gums around the teeth on pledgets of cotton wool. Follow this with hydrogen peroxide similarly applied. Black chromic oxide is immediately produced and should be allowed to remain in the tissues for about a minute, following which a further hydrogen peroxide mouth-wash should be given. These pain-relieving measures can be usefully supplemented by penicillin, either systemically or locally, for three or four days.

External otitis.

This condition is predisposed to by wax in the ears and by tropical climate which produces a sodden macerated meatal skin. It results more frequently from infection from a dirty towel than from bathing. Those who wear head-phones are particularly liable to suffer.

Treatment —In the tropics, wireless telegraphy personnel should have their ears inspected every week, their external meatuses cleared of wax, and 70 per cent. spirit drops instilled. Bathers should be instructed to drain the water out of their ears by tilting the head to one side, and in those predisposed to infection, vaselined cotton wool can be inserted before bathing. Other people's towels should not be used.

When the condition is established, gently syringe the meatus with 2 per cent. bicarbonate solution to remove debris, and dry carefully with a cotton wool swab. Then apply drops of 10 per cent. Mild Silver Protein B P C (Argyrol) in spirit. For the more acute case with constitutional symptoms, pack the ear with gauze wick moistened with 8 per cent. aluminium acetate solution, keeping it moist from a drop bottle for 24 hours, after which it should be changed. Hydrogen peroxide and fomentations should not be used.

PSYCHIATRIC CASES AT SEA

and hysteria is much the same
serious psychotics are concerned

special circumstances obtain. Thus, the means of suicide and manslaughter are ready at hand, panic is easily engendered and vitally important machinery may be tampered with.

In handling cases of acute psychosis the ship's doctor should bear in mind the following points:—

(1) Acute mania is sometimes the presenting symptom of heat-stroke (*see page 339*) and severe cerebro-spinal meningitis, but if these two diseases are thought of, mistakes are unlikely to be made. •

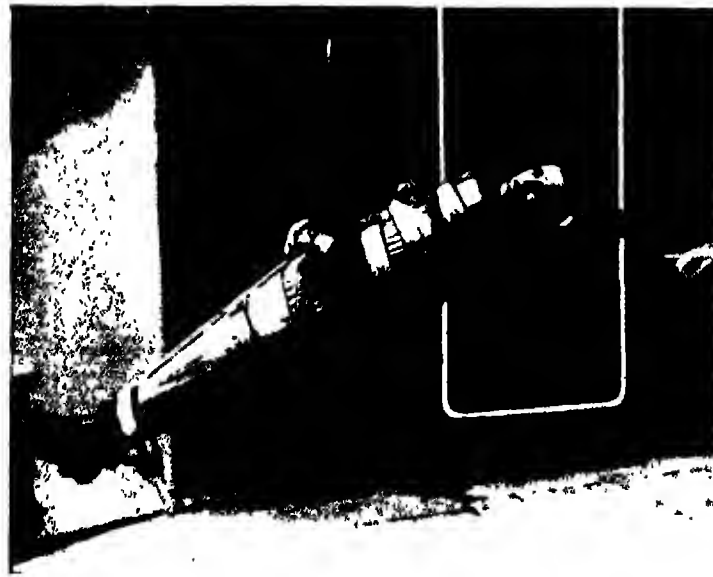


FIG. 42

The Neil-Robertson stretcher.

(2) Having decided that the patient is undoubtedly mad, the exact diagnosis does not matter very much. The problem is a short term one, namely, how best to get the patient to a port in a reasonable state of nourishment or without having done himself or others harm. Any form of treatment which will bring this about is justifiable, but when forcible restraint is used, medico-legal points arise (*see page 383*).

(3) The most dangerous period as regards suicide is in the *recovery* stage of acute depression. Most suicides are surprises. It is not true, however, that those who talk of suicide never commit it.

Treatment.—Put the patient in a spare cabin devoid of removable fittings, search him thoroughly, and have him watched night and day. See that he gets adequate nourishment, even if

this involves forced feeding. To control extreme violence, use a strait-jacket, a Neil-Robertson Stretcher (Fig. 42), or roll the patient in a sheet secured with safety pins or stitches.

In severe manic cases hyoscine hydrobromide 0.6 mg. (gr. $\frac{1}{100}$) and morphine sulphate 20 to 30 mg. (gr. $\frac{1}{3}$ to $\frac{1}{2}$) subcutaneously are often necessary. Sedation can be kept up with paraldehyde 5 to 10 ml. intramuscularly or amylobarbitone, 180 to 360 mg. (gr. 3 to 6) in capsule. In milder cases Nembutal (pentobarbitone) 100 to 200 mg. (gr. $1\frac{1}{2}$ to 3) or sodium barbitone (Medinal) 0.6 G. (gr. 10) are useful.

MEDICO-LEGAL PROBLEMS AT SEA

It is difficult to deal briefly with these, and in many cases definite rulings have not been laid down. Certain basic principles, however, are stated below, much help in compiling these having been obtained from A. V. Elder's *Ship Surgeon's Handbook*.

(1) Status of the doctor.

The word of the master is law, and the doctor must therefore obey his orders. If there is disagreement, the doctor must state his case in writing, and the legality or otherwise can be established later in harbour, the matter being laid before the owners, agents, or British Consul. If, for example, there were differences of opinion as to when a sick man was fit to resume duty, the master could have the final word, though it is extremely unlikely that he would interfere where a medical subject was in question. Again, the point might arise while embarking third class passengers at the gangway. Thus, Elder rejected a case of advanced locomotor ataxia with incompetence of the sphincters, while the agents urged acceptance on the plea of loss of passage money. In a case of this kind the master might have the last word and could over-rule the doctor, though again he would be extremely unlikely to do so.

(2) Fees.

The legal position is that the doctor must treat members of the crew without fee, but passengers, gratis. Some lines treat passengers, but allow acceptance of fees to limit fee-taking to illnesses requiring voyage. In practice this is very

difficult to decide, and in any case if the patient will not pay there is no effective machinery to make him. As Elder says, the doctor has no time to bother about proceedings for recovery and would probably be looking out for another ship if he did. Bad debts will inevitably be incurred at sea as on land. While in port in Great Britain and Northern Ireland officers and crew are under the National Health Service.

(3) Conflicting duties.

In the ship, the doctor has three loyalties:—

- (a) To the ship's company as a whole. Here he is acting in the capacity of Medical Officer of Health, with right of entry into all cabins and authority to advise detention of the ship in quarantine.
- (b) To the shipowners and to the Board of Trade. They will require records of sickness and work done, at any rate in respect of third class passengers and crew.
- (c) To his patients, in respect of professional secrecy. The following incident illustrates the above points (Elder):—

"A North Atlantic surgeon, whose ship was laid up, took a post as locum tenens in the Midlands. During his time there he saw and treated a patient for epilepsy. Some six months later, while examining passengers on embarkation, he recognised his late patient among them. Epilepsy being on the prohibited list, his position was awkward, to say the least of it. He allowed the passenger to embark, watched him during the voyage across, and as no fit occurred, he said nothing. The sequel took place a few months later. As a result of an epileptic seizure, the victim was taken to the immigration station at Ellis Island, where he admitted his complaint, and added that the surgeon in the ship in which he had crossed was quite aware of it, having treated him for it previously. The immigration authorities took the matter up with the steamship company and the surgeon. His attitude was, that as his knowledge of the patient's complaint was gained quite apart from his employment in the steamship company's service, or on the American route, he therefore considered it as 'privileged' and no part of his duty to report it officially. Whereupon the patient was deported and the matter dropped."

(4) Forcible restraint of lunatics.

If possible, obtain a second and independent certificate, inform the master, and make an official entry in the log book. If the patient is landed, the reason for this should be clearly stated on the certificate, as normally it is an offence to land a passenger at a wrong port without his consent.

(5) Accidents.

Full and careful records should be kept of all accidents, as litigation is often a sequel. If the surgeon has to continue treatment for an accident sustained before embarkation, a statement

will be asked for at the end of the voyage. Elder recommends a certificate stating that the holder has been treated between certain dates and nothing more. This will avoid discrepancies.

(6) Alcoholism.

Drunkenness is an offence under the Merchant Shipping Act, and punishment can be inflicted by the master. The doctor will be called upon to state whether a man is drunk or not, and usually a definite opinion has to be given—"drunk or sober," no intermediate condition being recognised.

Chronic alcoholism is a more difficult problem as it is not a crime, and the doctor can take no definite action unless the offender becomes a patient, when temporary abstinence can be enforced. (Incidentally, when a responsible officer drinks excessively, putting him sick is often the best course). If a chronic alcoholic becomes a public nuisance and passengers complain, it is the master and not the doctor who must take action. Although it will appear from this that pitfalls in the doctor's path are many, yet in practice they are usually avoided by a little tact and common sense.

C. A. CLARKE.

CHAPTER XXI

Medical Emergencies in the Air

WHILE the emergencies of flight may be attributable to failure in airworthiness of the aircraft they are mostly due to physiological and psychological factors involving the crew. If we include pilot error as well then most emergencies can be put down to human failure.

Modern air travel, with its complex requirements, may make the provision of adequate airworthiness increasingly difficult. Flight at high altitude, with its intrinsic potential dangers, has done much to increase this complexity. However, improvements in crashworthiness may yet prove to be the most fruitful source of saving of life since for a given expenditure in weight of an aircraft, such improvements would save more lives than would an increase in airworthiness alone.

True medical emergencies occur but rarely in flight. This does not, however, absolve the medical man from having knowledge of their nature, and he can aid greatly in the provision of proper advice to the intending traveller. From the military point of view, the medical officer dealing with flying personnel must of course have adequate knowledge of the environment of flight and the causes of physiological and psychological failure. He must also know the principles of casualty air evacuation and the emergencies that may arise. Many civilians are now employed as flight observers in government research establishments and aircraft firms and the medical officers engaged to safeguard their health must also know the nature of the emergencies to which they are most commonly exposed.

EMERGENCIES DUE TO ALTITUDE

These may be caused by anoxia or decompression. Modern aircraft can be classified as either unpressurised or pressurised. In the military field, unpressurised aircraft are normally those used in training, short range transport, and anti-submarine rôles. In the civil field many short range aircraft are still unpressurised whilst all inter-continental aircraft are pressurised.

The mechanics of pressurisation are relatively complex, but the general physiological principles, on which they are based, are clear. Sufficient pressures should be maintained to hold the cabin altitude at 8,000 feet (245 metres) no matter at what altitude the aircraft is flying. The Wet and Dry bulb temperatures of the cabin atmosphere must be maintained at comfort levels and a suitable proportion of fresh air to recirculated air admitted to prevent "stuffiness," the perception of odours and for general air hygiene. Military pressurised cabins may, of course, have different requirements.

Anoxia.

Lack of oxygen or anoxia is due to the decrease of barometric pressure which accompanies any gain in altitude. According to the standard International Convention of Aerial Navigation tables, the barometric pressure of 760 mm. Hg. at ground level (14·7 lb./sq. in.) is reduced to approximately one half at 18,000 feet (5,500 m.), one third at 27,000 feet (8,200 m.), one quarter at 34,000 feet (10,400 m.), and one fifth at 40,000 feet (12,200 m.).

Since the atmosphere is composed of 21 per cent. oxygen and 79 per cent. nitrogen and other inert gases, reduction in barometric pressure is reflected by a fall in the partial pressure of oxygen in the inspired air. Hence the partial pressure of oxygen in the inspired air at 18,000 feet (5,500 m.) is 79·5 mm. Hg. (or half the ground level value of 159 mm. Hg.).

The alveolar air is remarkably stable in composition at ground level. It is always saturated with water vapour at body temperature and in addition to oxygen and nitrogen, contains carbon dioxide. The average value of the partial pressure of oxygen in the alveolar air at ground level is 102 mm. Hg., and the fall in the partial pressure of inspired oxygen is also reflected in the alveolar air although modified by the occurrence of hyperventilation with its due effect upon the functional residual air. Thus the average alveolar partial pressure of oxygen of a normal resting young man at 18,000 feet (5,500 m.) is 35 mm. Hg.

The partial pressure of alveolar oxygen saturates the arterial blood to a value of about 97 per cent. at sea level, provided that the diffusion constant of the lungs is normal and that there is no large venous shunt. Owing to the S shape of the dissociation curve of oxyhæmoglobin, the fall in the alveolar partial pressure of oxygen has little effect on the oxyhæmoglobin saturation at altitudes below 10,000 feet (3,050 m.) in normal subjects. This is, of course, of great economic value to civil aviation since by

international statute the carrier corporations need only provide therapeutic oxygen for 10 per cent. of the passengers.

With further increase in altitude, the fall in tension of oxygen in the arterial blood stimulates the carotid and aortic chemoreceptors and hyperventilation commences in normal subjects at altitude of between 12 - 15,000 feet (3,660 - 4,000 m.). At the same time the fall in the oxyhæmoglobin saturation produces effects which can now be assessed objectively. The table* (Fig. 43) relates the various factors.

<i>Cabin Altitude</i>		<i>Alveolar Oxygen pp.</i>	<i>Arterial Oxy- hæmoglobin Saturation</i>	<i>Physiological Effect</i>
1,000 ft.	Metres	mm. Hg.	per cent.	
0	0	102	97	Nil
5	1,530	82	93	Nil
8	2,450	65	91	Nil
10	3,050	61	89	Slight handicap
18	5,500	38	71	Appreciable handi- cap
22 } to } 25 }	6,730 } to } 7,630 }	30 } or } below }	60 or below	Unconsciousness

FIG. 43—The effects of altitude.

SIGNS AND SYMPTOMS.—The effects of oxygen lack are insidious and only the well trained observer will have sufficient powers of introspection to realise that he is anoxic, but even he may have such impairment of judgment that he cannot analyse the cause of his disability. Thus any mechanical warning system which is not automatic but which demands that the traveller performs some action cannot be fully efficient under any circumstances. Excepting the effects of mild anoxia on the elderly and infirm, under normal circumstances the modern traveller has a warning of impending anoxia in the sudden release of cabin pressure consequent upon failure of the cabin.

Subsequent effects then depend upon five factors:—

- (i) The altitude.
- (ii) The rate and speed of descent.
- (iii) The degree of activity of the passenger.
- (iv) The final altitude for continuation of flight.
- (v) The state of health of the passenger.

Up to an altitude of 25,000 feet (7,630 m.) the first and second factors may be treated together and it can be supposed that descent to below 15,000 feet (4,000 m.) or below can be accomplished in 3 to 5 minutes. In the normally fit individual, such a degree of anoxia will not constitute a real emergency. Exceptions, however, can be found in the elderly and infirm, or in certain normal individuals who are unduly susceptible to anoxia, and become unconscious rapidly from syncope following upon vasodilation of the blood vessels of the muscles. Less than 1 per cent. of normally fit individuals will behave in this manner.

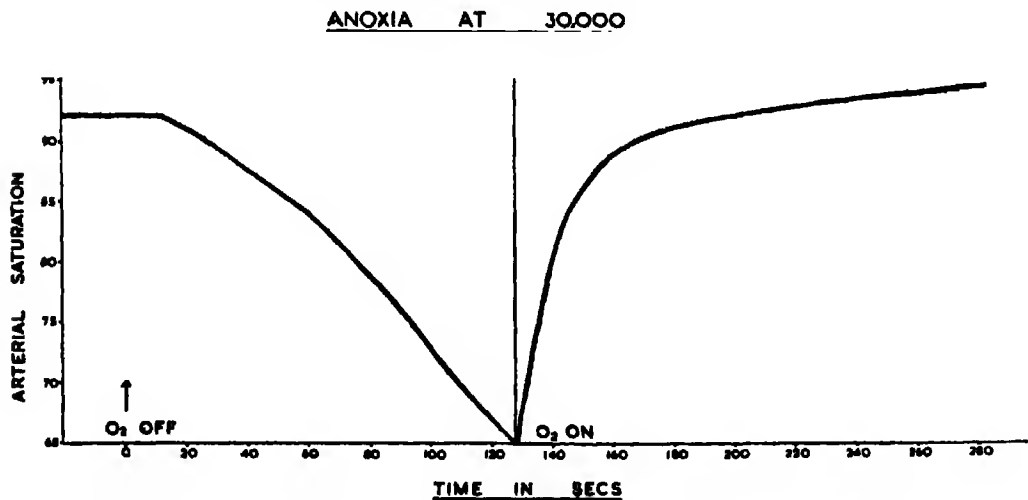


FIG. 44 —Time for desaturation and resaturation of arterial blood.

At altitudes about 30,000 feet (9,200 m.) anoxia following upon decompression constitutes a major medical emergency. The onset of unconsciousness is rapid and at altitudes above 35,000 feet (10,650 m.) is likely to be complete. At 40,000 feet (12,200 m.) consciousness is lost in 20 to 30 seconds following upon decompression and this time is due to the rapidity of desaturation of the arterial blood which is great even at slightly lower altitudes (Fig. 44).

The degree of activity of the passenger at the time of the decompression is of some importance, particularly within the altitude range of 25,000 feet to 35,000 feet (7,630 - 10,650 m.). If away from his seat, he may well become unconscious from the muscular activity involved in reaching it particularly if the aircraft is descending in a steep dive. A passenger unconscious on the floor is potentially at greater risk than in his seat, and in addition, may affect other persons adversely if they attempt to aid him without using their oxygen equipment.

The final altitude for continuation of flight may be of considerable significance particularly in the case of intercontinental travel in turbine-engined aircraft. If this altitude is maintained above 15,000 feet (4,500 m.), the load on the circulation of ill or elderly passengers would undoubtedly cause symptoms unless adequate oxygen could be given continuously.

Anoxia in the elderly, ill or infirm traveller may cause medical emergencies of the most important kind. Whittingham states that about 90 passengers a year require medical attention during flight and since most of these episodes occurred in flights of six hours or more, anoxia very probably played an important part. Most of those taken ill suffer from cardio-vascular disease. The blood in the coronary sinus is almost completely desaturated under normal conditions and therefore any degree of anoxia must throw a load on a damaged heart, since vasodilation must ensue to maintain sufficient oxygenation in the myocardium.

Diseases causing respiratory symptoms require careful assessment. Any cases with a large venous shunt, *i.e.*, any degree of desaturation particularly under moderate exercise, tolerate anoxia badly. There is little known on the allowable limits but cases have been investigated with vital capacities of 1,200-1,400 ml. and maximum breathing capacities of 40 to 50 litres who desaturate on standing at 8,000-10,000 feet (2,450-3,050) breathing air. There is probably danger wherever the functional residual capacity is so small that the tidal air forms a large portion of the alveolar air. Anoxia has recognisable risks in anæmic travellers especially if the hæmoglobin is below 50 per cent. In general it can be stated that very little is known of the effects of severe anoxia in the elderly and unfit. It can be assumed that decompression above 30,000 feet (9,200 m.) might prove fatal in many of them.

DIAGNOSIS.—The circumstances attending the emergency, the previous history and the nature of the symptoms such as breathlessness should suggest the diagnosis in all cases. It should not be forgotten however that the anoxia may only have been a precipitating factor—as for example in an anginal attack. The administration of oxygen may serve to differentiate since the less severe cases and the respiratory and anæmic cases tend to recover after 5-10 minutes at the most on adequate oxygen.

Treatment.—Normally therapeutic oxygen is available in most passenger carrying aircraft. Such oxygen sets usually last for a

maximum of one hour or less depending on the flow used. A light-weight rebreathing mask is generally used with flows of either 3.5 L/min. or 10 L/min. The higher flow should only be used in very severe cases or if decompression has occurred at altitudes above 25,000 feet (7,630 m.). The mask can be altered to fit babies, young children or adults.

If possible, the aircraft should descend so that the cabin altitude is not greater than 5,000 feet (1,530 m.). However, if the emergency is general and due to complete loss of cabin pressure, the captain will descend immediately and any confusion can be remedied at a lower altitude. Emergency oxygen sets should be issued at the first opportunity to those passengers who most require them. It is absolutely necessary for the passengers to remain as calm as possible and inactive in their seats. If any passengers thereafter remain ill, instructions can be relayed to the aircraft from the nearest ground station. A first aid set with cardiac stimulants and sedatives is carried.

SEQUELÆ.—These may only be observed in severely ill patients. Most of the available information relates to military passengers who have been exposed to severe anoxia for some time. In these cerebral dysfunction is the dominant after-effect, but its extent is variable, and depends upon other circumstances such as cold and probably individual sensitivity. For example, unconsciousness for 2 - 3 minutes breathing air at 35,000 feet may result in coma for as long as 12 hours, or, merely marked disorientation. Sometimes death occurs but this is uncommon in normally fit young subjects. An abnormal electroencephalogram may remain for days but a normal clinical recovery is made in most cases.

The duration and degree of anoxia necessary to cause death is extremely variable but military experience has shown that a fatality must never be assumed and that artificial respiration with therapeutic oxygen should be continued for as long as possible.

Physical Effects of Decompression.

The consequences of decompression depend both on rate and degree of pressure change. The barometric pressure at 8,000 feet (2,450 m.) is 10.9 lb/sq. in. and hence even in modern aircraft most travellers are routinely exposed to a pressure change of 3.8 lb./sq. in. In travellers with acute blockage of the Eustachian tubes from nasopharyngitis or similar conditions, such a total pressure change is sufficient to cause acute pain. Normally how-

ever, the rate of pressure change is sufficiently slow to avoid trouble except in inexperienced individuals. Most symptoms can be avoided by judicious swallowing or breathing out with the mouth closed and nose pinched immediately a pressure change can be felt upon the ears. In the case of forced descent after decompres-



FIG. 45

Normal subject after barium meal at 35,000 feet (10,730 m.).
Striking distension of the cæcum and ascending colon.

sion, the pressure change is usually greater; in piston engined aircraft it may be 4.5 lb./sq. in., and it must be assumed that, in a significant number of passengers, pain will ensue. In turbine-engined aircraft the pressure change could be as great as 7 lb/sq. in. This will not only cause pain but also severe congestion of the middle ear with temporary loss of hearing and rupture of the tympanic membrane in some cases. Although not constituting a danger to life, such effects may well reinforce the effects of other factors in decompression, such as anoxia.

The volume of gas in the abdominal viscera at ground level is normally between 500 and 1,000 ml. It does not cause symptoms if the rate of pressure change is slow and steady up to altitudes of 25,000 feet (7,630 m.) (a total pressure change of 9.2 lb./sq. in.). However, following upon rapid decompression from 8,000 feet (2,450 m.) to above 30,000 feet (9,200 m.) (Fig. 45), gas expansion commonly causes severe abdominal colic and syncope in some subjects.



FIG. 46

Right-sided pneumothorax at ground level.

It must be emphasised that all studies in this field have been made on fit young males and nothing is known about the reactions of the elderly, the unfit or the female sex.

In certain military aircraft such a pressure change may occur in one or two hundredths of a second and then rupture of the elastic tissue of the lung must ensue. Owing to the large volume of the cabin of transport aircraft the loss of pressure lasts five seconds or more and this is not a hazard in travellers with normal lungs. Serious effects would undoubtedly ensue, however, in patients with

localized cavities. Experiments with fit young adults have shown that decompression from 8,000 ft. to 35,000 ft. (2,450 to 10,650 metres) in 0.09 seconds proved completely innocuous. In the disasters to the Comet aircraft in 1954, the primary failure in the pressure cabin involved the loss of a maximum area of about 160 sq. ft., which resulted in a time of decompression of slightly greater



FIG. 47

Same patient as in Fig. 46 at altitude of 9,000 feet (2,743 m.), showing increased collapse of right lung and mediastinal displacement.

than 0.06 seconds. The chief results were severe fatal injuries to the skull and trunk due to violent displacement by the great loss of energy which could be likened to the explosion of a 100-lb. bomb. Any effects upon the lungs were undoubtedly masked by the impact with the sea, after a period of free fall.

Patients with artificial pneumothorax or pneumo-peritoneum should not under any circumstances be exposed to altitudes greater than 8,000 feet (2,450 m.) and preferably 5,000 feet (1,530 m.). Embarrassment of cardiac and respiratory function develops if the

pressure change is of the order of 3.5 to 4 lb./sq. in. (Figs. 46 and 47).

Decompression Sickness.

Maintenance of flight above 30,000 feet (9,200 m.) after decompression is a current problem of turbine-engined military aircraft



FIG. 48

Gas in the knee joint. Exposed for three hours at 40,000 feet (12,200 m.).

capable of flights of long duration. It is not yet a problem of civil aviation. After any possible consequences of anoxia have been resolved, there remains this problem of decompression sickness which is related to the elimination of nitrogen from the body.

A normal fit young man, of average body build and weight, has about 1,100 ml. of nitrogen dissolved in his body fluids and tissues at a tension of 574 mm. of mercury. The quantities in

different tissues varies with their respective coefficients of solubility. If the pressure of the environment is reduced, not only will there be a gradient of nitrogen pressure between the body tissues and the lungs, nitrogen being exhaled, but also the nitrogen in solution in the tissues, saturated at sea level pressure, will be in a state of supersaturation and may theoretically come out of solution and form bubbles. For any given rate and value of pressure change, this tendency to supersaturation will be greatest in those tissues with the least blood supply and the greatest dissolved nitrogen content, *i.e.*, fatty tissues.

The physical explanation of the formation of bubbles is complex and not only involves the differential pressure between the dissolved nitrogen and the total barometric pressure, but also the hydrostatic pressures in the tissues of fluids, surface tensions and possibly the deformation pressures of the growing bubble. These bubbles may then give rise to the signs or symptoms of decompression sickness, either by direct pressure or indirectly. Tissue damage may occur with more generalised effects, but although bubbles have not been seen in man, gas pockets within fascial planes or joints have been observed (Fig. 48). Bubbles have been found on many occasions experimentally in animals and there is as yet no better explanation of the cause of decompression sickness.

SIGNS AND SYMPTOMS.—The commonest symptom is pain in joints, particularly the shoulder, knee, wrist and ankle and in those muscles subject to most activity. The pain, called "the bends," is most often mild but may develop, with maintenance of altitude, into a severe visceral type, ill localised, and incapacitating. It is not abolished by morphine but is abolished by descent below 25,000 feet.

Other symptoms and signs may appear after the onset of pain although infrequently the first severe symptom may occur without preceding pain. Neurological signs such as paralysis or fits are rarely seen at altitude, but in subjects susceptible to migraine, severe incapacitating headaches with dimness of vision, scotomata or hemianopia and fortification spectra have been noted with sufficient frequency to encourage the belief that decompression procedures might possibly be used to study the mechanism of migraine.

In passengers, as well as aircrew, the most important effects are related to the respiratory and circulatory systems. Retrosternal

pain ("chokes") may occur particularly during deep breathing. There is a dry hard cough and perhaps cyanosis. Occasionally the pain is of sudden onset and localised laterally. If recompression be not undertaken immediately the condition can progress to collapse. Typical syncope can also occur, characterised by vague nausea and sweating progressing swiftly to a catastrophic fall in blood pressure and unconsciousness.

The relative frequency of these symptoms is of some interest; in 610 two-hour tests at an altitude of 37,000 feet (11,300 m.), at the R.A.F. Institute of Aviation Medicine, the percentage of forced descents for "bends" was 11.6, for syncope 4.6, for "chokes" 2.8, and for other causes 2.3. Minor symptoms such as uneasiness, inability to concentrate, skin rashes, skin oedema are also observed. Paræsthesiæ of all types are common. Post-decompression effects, though infrequent, are of great importance. Ocular signs and symptoms may persist for several hours after exposure and paresis has been known to last 24-48 hours. Occasionally more severe neurological after-effects have been encountered with apraxia, gross ataxia, aphasia, mirror writing, extensor plantar responses and epileptiform convulsions. The EEG may be abnormal for some days. The prognosis in these cases is always good.

More serious are the cases of apparent recovery from "bends," "chokes" or syncope which develop what is now termed post-decompression shock. This may occur up to four hours after exposure and merits close observation in any subject who complains of nausea, headache or vague visual disturbances within this period. The blood pressure is usually unstable and repeated hæmatocrit readings may reveal hæmoconcentration. There may also be signs of pulmonary oedema or pleural effusion. A small number of these cases are progressive so that hospitalisation may be required at an early stage. The whole picture is that of acute secondary shock, but the ætiology of the condition is as yet unknown.

The effect of exposure of elderly persons to prolonged decompression at these altitudes is also unknown, although the increase in the time of diffusion of gases through the lungs with increasing age must point to a greater frequency of severe effects. For a stay of one hour at 37,000 feet (11,300 m.) the occurrence of severe symptoms in the age group 35-47 years was five times as great as in the age group of 18-25 years.

DIAGNOSIS.—The symptoms of decompression sickness normally do not develop immediately upon loss of pressure within a cabin and can usually be differentiated from oxygen lack. The average time is between 30 and 60 minutes but the scatter is large. Recompression below 25,000 feet (7,630 m.) will usually result in return of consciousness provided that oxygen be given, whereas the administration of adequate oxygen about 30,000 feet (9,200 m.) might not be efficacious if the syncope is due to decompression sickness. No statistical information is available as to progress should the altitude be maintained.

Treatment.—Recompression remedies the condition in the great majority of cases.

Prevention is all important and thus the cabins of high altitude transport aircraft must be designed for absolute integrity of pressure. In military personnel, where this may not always be possible, preselection of the more resistant men can be ensured by preliminary exposure in decompression chambers. The failure rates vary according to the exposure time, and the altitude; 8 per cent. of subjects fail if exposed to 35,000 feet (10,650 m.) for one hour, and 20 per cent. at 40,000 feet (12,200 m.) for the same duration.

Denitrogenation can be satisfactorily accomplished in selected instances as in test pilots, but has not been found to be satisfactory in military personnel under operational conditions. If the nitrogen in the inspired air is replaced by oxygen at ground level, about 80 per cent. of the dissolved nitrogen is replaced by dissolved oxygen if breathed for three hours. This will almost certainly protect sensitive subjects since, presumably, no bubbles form on decompression as the oxygen is metabolised too rapidly. However, the remaining 20 per cent. of nitrogen is probably dissolved in inaccessible tissues with a very long half time of excretion, and to give full protection for a stay of any duration at altitude a very long time of preoxygenation is necessary.

The treatment of cases of post-decompression shock must be instituted as early as possible. A case with a hæmatocrit reading of 65 per cent. 30 minutes after decompression has been observed, hence the necessity for close observation in the preliminary period. Although the patient may remain rational, the administration of adequate fluid intravenously and oxygen should be instituted before removal to hospital if possible, but hospitalisation should under no circumstances be delayed. If the hæmoconcentration

proves to be progressive, cortisone or intravenous noradrenaline may be administered.

SEQUELÆ.—Apart from fatal cases of post-decompression shock, and cases of delayed recovery with neurological signs and symptoms, there are normally no sequelæ. This is in contradistinction to caisson disease in which gross neurological damage or changes in the architecture of the long bones are not uncommon (*see page 376*).

FITNESS FOR AIR TRAVEL

Cardio-vascular disease

Patients with cardiac lesions are apt to react badly to low oxygen tension, even at cabin altitudes below 8,500 feet (2,591 m.). If they travel by air, it should be in a pressurized aircraft; their movements in the aircraft should be limited and oxygen should be available. Those suffering from angina pectoris should have the condition adequately controlled and an electrocardiogram should be done to exclude infarction. Such cases can be accepted for air travel if the angina is provoked only by a brisk walk of about half a mile, and if attacks are not frequent. Cases of coronary occlusion should not fly unless they have been free of symptoms for at least six months.

Patients with valvular disease of the heart should not fly while the condition is acute, nor when it is chronic if there are signs of congestive failure or marked anæmia. Those with congenital heart lesions can be accepted for flights at 5,000 to 6,000 ft. (1,530 to 1,840 m.) if there is no marked veno-arterial shunt as shown by cyanosis, and if there is no heart block or severe hypertension resulting from coarctation of the aorta.

Cases of high blood pressure are acceptable provided they have flown before and there are no serious complications such as pulsus alternans, cardiac asthma, frequent headache, marked loss of weight, optic fundus change, gross albuminuria, or cellular casts in their urine. Those who have not flown before may be unduly upset by nervous tension, and should be given a sedative. Care should be exerted when the blood pressure at rest is of the order of 200/100.

Respiratory disease

Oxygen should be available whenever patients with respiratory diseases are being transported by air.

Each asthmatic case must be considered on its merits and it is well to ascertain whether there is any allergy to pyrethrum, as this is an ingredient of all insecticides used in aircraft. Milder asthmatics may even find their condition improved by flying at moderate altitude. Those who suffer with severe attacks, especially if there is a cardiac or renal basis, should not travel by air.

Patients with bronchitis and emphysema and with fibrosed lungs should not normally fly higher than a cabin altitude of 8,500 feet (2,591 m.). The decision as to whether they are fit for a long air journey rests on such points as the degree of dyspnoea, and whether there is offensive sputum or any cardiac lesion. They should move about as little as possible while in the air.

Lobectomy and pneumonectomy cases should not travel by air within three months of the operation. Their degree of breathlessness on exertion is a good guide to their fitness for the journey, but vital capacity of less than 2 litres or a low maximum breathing capacity may require the patient to remain seated whilst the cabin is at altitudes of 8,000 feet (2,439 m.).

Patients with active pulmonary tuberculosis should not travel by airliner unless their sputum is free from tubercle bacilli.

Gastro-intestinal disease

Those with active peptic ulcers should be warned against air travel, owing to the danger of gaseous distension causing hæmorrhage or perforation. Cases of colostomy are not acceptable, as the decreased atmospheric pressure and distension of the gut leads to passage of fæces and flatus with consequent odours, which are objectionable to fellow-travellers.

Post-operative cases

Patients who have had an operation such as appendicectomy or herniotomy should not travel within 10 days of the operation, and the cabin altitude should not be over 8,500 feet (2,591 m.). Anti-air sickness remedies should be prescribed.

Diabetic and elderly passengers

Diabetics should not travel by air unless the condition is fully controlled. Only mild and moderate cases which are stabilised by diet or insulin and have no obvious cardiovascular disease, are acceptable for long-distance air travel. They should be capable of administering their own insulin.

Elderly persons travel well at cabin altitudes up to 10,000 feet (3,050 m.), provided they are free from organic disease. It is advisable for those over 65 who are undertaking a long flight for the first time, to have a medical examination to exclude anæmia, cardiovascular disease, and nephritis. In cases of doubt an electrocardiogram is necessary. Obese persons, even if they are young, should be examined carefully for cardiovascular disease.

Pregnancy

There are no contra-indications to a pregnant woman flying providing the aircraft is pressurized and that the pregnancy is not advanced beyond the eighth month. If liable to vomit she should take an airsickness tablet before the flight, and should, if possible, have a backward-facing seat, so that the belt need not be very tight.

EMERGENCIES IN CASUALTIES OR INVALIDS

Airsickness

Airsickness in fit individuals cannot in itself be regarded as a true medical emergency, even when the victim is utterly prostrate, but in certain cases it might precipitate a true emergency.

It is caused by repetitive movements of the aircraft. The otolith responds to linear gravitational accelerations in the vertical plane and the semi-circular canals to angular acceleration. There is evidence that although stimulation of the hair cells of the utricle by the otolith is chiefly responsible, accelerations affecting the semi-circular canals reinforce the effects. In the stage of nausea, pallor and sweating, there are few systemic effects, apart from hyperventilation. The chief danger lies in the occurrence of intractable vomiting. This must be particularly avoided in advanced pregnancy and in patients with peptic ulcers, diabetes and those who have had a recent abdominal operation, or a head injury. In addition, the motion may precipitate attacks of biliary or renal colic.

Treatment.—Prevention is desirable since vomiting once started cannot usually be aborted by any known medication. "Hyoscine hydrobromide 0.65 mg. (gr. $\frac{1}{100}$) half an hour before take-off is still the drug of choice. The anti-histamine drugs may be used if suitable in individuals who prefer them from past experience. A combination of hyoscine 0.65 mg. (gr. $\frac{1}{100}$) and diphenhydramine (Benadryl) 25 mg. is particularly effective. Selected patients

should lie supine near the centre of gravity of the aircraft, and in particularly rough weather it is advisable to fix the head as comfortably as possible.

Epilepsy and Mental Disorders

A number of examples of loss of consciousness from various causes in flying personnel were collected during the war. In about 25 per cent. an abnormal electroencephalogram was recorded. Whilst fear, hyperventilation or low blood sugar may have been the precipitating causes, it was felt that some may have been of reflex origin from the activity of flying. It is not considered that this type of case may be encountered in passengers under normal circumstances, but attacks may be precipitated by acute fear in any emergency such as ditching or crash landing or temporary loss of control in gusts. It is also thought that some epileptics might react badly to travel in helicopters with blade frequencies of 8-12 cycles per sec. causing visual stimulation.

Miscellaneous emergencies

Strict observation of the rules constructed by Whittingham and other medical directors of airline companies should obviate other emergencies. First-aid kits are carried in all commercial aircraft in accordance with I.C.A.O. regulations and include antiseptics, analgesics, narcotics, a cardiac and respiratory stimulant and a remedy for burns. Stewards and stewardesses normally undergo an adequate training in first-aid and are in charge of the first-aid outfit. Any doctor or nurse required to accompany the patient must be engaged by the passenger. The doctor should see that all necessary drugs and appliances are present and that the patient fully understands any action necessary en route. Instructions should be written clearly and this should particularly apply to long journeys where travelling involves alteration of time schedules.

MEDICAL ASPECTS OF CRASHWORTHINESS

Improvements in crashworthiness would save more lives than all those lost from pure medical emergencies during flight. The forced landing casualty can be regarded as a true medical emergency except when a crash occurs at or above a normal cruising speed. Controversy still exists as to the best internal layout of an aircraft for avoiding injuries in crash landings at the normal approach speed.

Since the original British researches which led to the introduction of backward facing seats, there have been no fatal casualties to passengers in aircraft of R.A.F. Transport Command fitted with such seats. There have been fatal casualties in civil aircraft of similar basic design and in accidents of similar degree of severity, the difference lying in the use of less strong seats facing forwards and with lap straps for restraint.

There is abundant proof from experimental work to suggest that most fatalities result from fractures of the skull and that other injuries are to some extent subsidiary. The major cause of death is not rupture of viscera and large blood vessels from the snubbing action of safety belts, except when there is underlying disease. Such injuries generally occur when belts rupture or seats tear away.

In forward facing seats the incidence of injuries, apart from the overall severity of the crash, are dependent upon the structure of the seat in front of the individual and the pitch or distance between similar points on the seat.

Since the body flexes to a remarkable degree with severe loads and when restrained by a lap strap, and since the pitch in modern aircraft is never sufficiently great to avoid striking the head, two protective actions only can be recommended. Firstly, the trunk can be flexed and the head rested in the crook of the arm on the back of the seat in front. Secondly, the seat can be laid back and the feet braced on the supports of the seat in front.

No assessment can be made of the relative efficiency of these two methods, but both suffer from the marked disadvantage of applying additional loads progressively down the line of seats so that the forward seat may fail with a resultant disastrous effect.

Backward facing seats do not suffer from any of these disadvantages. The load is not applied through a narrow belt but evenly over the back. Good protection can also be given to the head by a stressed head rest in the event of the aircraft overturning.

In all these aircraft there are structural limitations to improvement. In the fuselages of present types of civil aircraft the limits of strength is a load ten times gravity (10 g.), whereas in the rear portion the strength is of the order of 20 g. It is clear, therefore, that even if all seats were stressed to only 10 g., very substantial protection could be achieved provided that they faced backwards.

W. K. STEWART.

CHAPTER XXII

Ophthalmic Emergencies

FOREIGN BODY IN THE EYE

NOTE particularly the mode of entry. Some particles are simply carried in by a current of air and are usually lightly adherent. Others enter with force as occurs in hammering or grinding; these are often firmly embedded and may actually penetrate the globe.

If photophobia makes examination difficult, instil a drop of 1 per cent. amethocaine hydrochloride (there is no contra-indication to this way of facilitating examination in any type of painful eye). Illumination is best provided by a strong spotlight directed obliquely.

- (i) Explore the corneal surface carefully, sector by sector, remembering to direct the attention to the transparent dome and not to the deeper and more obvious iris markings.
- (ii) Examine the bulbar conjunctiva and lower fornix.
- (iii) Evert the upper lid (Fig. 49). A foreign body is frequently lodged in the tarsal sulcus which runs some 2 to 3 mm. from the free margin of the lid.

If no foreign body is found, suspect:

- (i) Spontaneous exit leaving a small corneal abrasion. The site of this can often be found by "staining." An area denuded of epithelium shows up as a green spot after instilling a drop of 1 per cent. fluorescein (*see page 43*) and washing out the excess with 1 per cent. boric acid or physiological saline.
- (ii) An inturning eyelash.
- (iii) Penetration of the globe. The entry must be forcible for this to occur but the possibility is frequently forgotten with resulting loss of an eye.

Removal of foreign body.

A foreign body under the upper lid is best removed by everting the lid and wiping it off with a damp cotton wool stick or corner of a clean handkerchief (Fig. 49).

Corneal foreign body (Fig. 50).



A.

B.



C.

D.

(Photographic Department, St. Bartholomew's Hospital.)

FIG. 49—Eversion of the upper lid.

- A. The patient must look down and remain looking down throughout the operation. The match is placed just above the upper lid crease which marks the upper border of the tarsal plate.
- B. The lid is drawn away from the globe by the lashes.
- C. The lid is drawn forward and upwards around the end of the match which is gently depressed at the same time.
- D. The foreign body lying in the tarsal sulcus is removed by a cotton wool stick.

As fainting is an occasional complication during removal, the patient should lie on a couch. If this is not possible, the head must be kept back and rested firmly on a support. Three drops of 1 per cent. amethocaine are instilled at minute intervals. It is important to gain the patient's confidence before starting the removal by demonstrating that the procedure will be painless. This is done by lightly touching the bulbar conjunctiva (which is in any case the least sensitive part) and then the cornea.

The gaze is then directed upward to some suitably placed spot and an assistant focusses a spotlight obliquely on the cornea. Fig. 50 shows the method of holding the lids to prevent blinking and also how the hand holding the instrument is steadied by resting it on the patient's cheek. If the operator is right-handed, he should stand on the patient's right side for either right or left eye.

The choice of instrument used depends on the degree of adherence of the foreign body and the experience of the operator. Removal can frequently be effected with a damp cotton wool wick, but a spud will be necessary for more firmly embedded particles. The least traumatizing procedure is to use a pointed instrument such as a Bowman's needle or a large straight triangular surgical needle (these may with advantage be used in conjunction with a magnifying loupe). The point is introduced under the foreign body which is then lifted off. This procedure is not without risk of penetrating the cornea but practitioners who have frequent opportunity to practise the operation will find it the most effective method.

After removal instil one drop of Eye-drops of Penicillin B.P.C. and a drop of 1 per cent. homatropine. Keep the eye padded until healing is complete—this normally takes 24 to 48 hours. A sensation as though the particle is still present indicates that the area has not yet been covered by epithelium and staining with fluorescein will confirm this.

Complications of removal of a foreign body.

- (i) Extensive corneal abrasions. These usually result from repeated unsuccessful attempts to dislodge a particle which may have penetrated deeply. If the foreign body will not come away after one or two attempts, expert help must be obtained.

- (ii) Perforation of the cornea. A gush of aqueous and a shallow anterior chamber indicate this complication which fortunately is not usually so serious as might be expected. The attempt at removal is, of course, abandoned. Several drops of penicillin (Eye-drops of Penicillin B.P.C. which contain 2,500



(Photographic Department, St. Bartholomew's Hospital.)

FIG. 50

Removal of corneal foreign body.

The patient is directed to fix the gaze on some distant object. The left thumb retracts the upper lid to prevent closure during the operation. The hand holding the spud is steadied by resting the lower three fingers on the patient's cheek.

units in 1 ml.) are instilled and one of 1 per cent. atropine. The eye is lightly bandaged and the patient treated as for penetrating injury (*page 409*).

- (iii) Corneal ulceration. This is a later complication caused by infection of the traumatized area. It is most likely to occur if the eye is left unbandaged before the wound has become properly healed.

SPECIAL TYPES OF FOREIGN BODY

1. RUST RING.

A ring of very adherent brown stain often occurs around a steel or emery wheel particle. The parent foreign body is often quite easy to deal with, but the eye usually remains irritable until the rust ring has been removed. When the condition is recognised the case is best referred to an expert as blunt ended instruments are quite useless and scraping the area with a sharp needle or small dental burr is necessary.

2. GLASS

Glass particles are difficult to see although they may glint in a bright light. Even quite small particles may, however, be discovered by instilling 1 per cent. amethocaine and then running the ball of the little finger along the lower fornix and over the surface of the everted upper lid.

3. LIME. (*page 411*).

4. INDELIBLE PENCIL.

Chips of indelible pencil or aniline dye in any form set up an intense conjunctivitis. Remove any visible particles with cotton wool wick or fine forceps and instil pure glycerine at ten-minute intervals for two hours.

5. MULTIPLE PARTICLES.

Sand, dust, and other particles are best removed by liberal irrigations of the eye with a stream of physiological saline.

INJURIES CAUSED BY POINTED OR BLUNT OBJECTS

These injuries fall into two groups :

- (i) Those perforating or rupturing the globe. There is attendant risk of early intra-ocular infection or subsequent sympathetic ophthalmia. Intra-ocular foreign bodies come into this category but the penetration is more commonly caused by a scissors or other pointed object.
- (ii) Those causing contusion of the globe. Injuries of all grades of severity may be caused by rounded objects such as knuckles, tennis balls, and projections on furniture.

A sharp instrument may damage the eye without entering and a blunt instrument may cause rupture of the globe. Hence the history may be misleading. A careful examination is essential.

Method of examination.

- (i) Estimate the vision of the injured eye. Even when the lids are severely bruised they can be gently separated and the test applied. Vision is conveniently assessed by finding what size newsprint can be read or at what distance fingers can be counted. If good vision has been retained, many serious possibilities are excluded.
- (ii) Examine the lids, cornea and sclera for evidence of puncture wounds.
- (iii) Stain the cornea with fluorescein (*pages 43 and 403*) for evidence of abrasion (a common injury).
- (iv) Examine the anterior chamber, noting the depth and the presence or absence of blood.
- (v) Examine the iris, noting any displacement or distortion of the pupil and compare direct and consensual light reflex with the uninjured eye.
- (vi) Dilate the pupil with 1 per cent. homatropine and examine the lens, vitreous and fundus with the ophthalmoscope.
- (vii) X-ray the orbit if an intra-ocular foreign body is suspected. *On no account should an attempt be made to test the ocular tension.* Little information is gained thereby and prolapse of intra-ocular contents may be caused if perforation has occurred.

PENETRATING INJURIES**Signs of penetration of the globe.**

- (a) If the wound is in the cornea or corneo-scleral junction, the pupil is drawn up towards the wound and there is likely to be a prolapse of iris through it. This will appear as a small brown or black bead on the surface, often with a thread of fibrin or mucus attached. The anterior chamber may be shallow or absent.
- (b) If the wound is in the sclera, it is more difficult to decide whether penetration has occurred as subconjunctival bleeding usually obscures the wound area. The appearance of brown uveal tissue or the presence of vitreous will clinch the diagnosis.

Emergency treatment of penetrating injuries.

Pending removal to hospital, emergency treatment consists in combating possible infection by instillation of Eye-drops of Penicillin B.P.C. and applying a light pad and bandage.

General treatment consists in preventing further prolapse or intra-ocular hæmorrhage by keeping the head still and the body relaxed. Phenobarbitone is a useful sedative but morphine must be avoided as it may provoke vomiting. The patient should be taken to hospital in a lying position or, if this is not possible, sitting back comfortably with the head supported.

NON-PENETRATING INJURIES

The possible effects of a non-penetrating injury are:—

(i) Bruising of the lids.

This may make examination of the globe difficult although the visual acuity can usually be assessed.

(ii) Corneal abrasion.

This is a common injury. The extent can be ascertained by fluorescein staining (*pages 43 and 403*).

(iii) Intra-ocular hæmorrhage.

In the anterior chamber blood may be seen by the naked eye. In the vitreous hæmorrhage is revealed by absence of the red fundus reflex. Retinal hæmorrhages and also white patches of œdema (*commotio retinae*) at the posterior pole are seen by ophthalmoscopy.

(iv) Injury to iris and pupil.

The pupil may suffer traumatic mydriasis and the iris show tears at the pupil margin or at its peripheral attachment.

(v) Injury to lens.

A displaced lens may give rise to a tremulous iris and its rounded edge may be observed through the pupil on ophthalmoscopy. Cataract formation may occur in the ensuing weeks.

(vi) Retinal detachment.

This important complication, with its characteristic symptom of loss of part of the visual field, may not be apparent for some days or weeks after the injury.

(vii) Secondary glaucoma.

Severe pain in the eye, forehead or temple should arouse suspicion of developing intra-ocular tension. It is most likely to occur after intra-ocular hæmorrhage.

Treatment of non-penetrating injuries.—The swelling of even severely bruised lids soon resolves with a cold saline compress and firm bandage. Corneal abrasions are treated by 1 per cent. homatropine drops twice daily and padding the eye until healed. If any intra-ocular damage is suspected, the patient should be kept quietly in bed with the head supported on two pillows and the advice of an ophthalmologist obtained.

PHYSICAL INJURIES

Burns of the lids usually occur from accidents such as "blow-backs" from gas stoves, petrol explosions and the like (*see also page 431*). The skin of the lids and face may suffer any degree of burn. The eyes suffer corneal abrasions and may receive multiple foreign bodies which are either the products of the explosion or fragments of the patient's charred lashes.

Treatment.—Gently cleanse the lids with warm saline and remove dead skin with scissors. Clip the lashes almost to the roots; scissors smeared with petroleum jelly will be found helpful as the cut lashes adhere to the blades instead of dropping into the eye. Irrigate with warm saline to remove foreign particles and examine the cornea for abrasions after fluorescein staining. Instil penicillin drops and cover the closed lids with tulle gras dressings. Apply a light pad and bandage. Coagulants such as tannic acid on the skin of the lids are absolutely contra-indicated. The stiff plaque resulting may prevent closure of the lids and allow exposure of the cornea.

Exposure to ultra-violet light.

This occurs in arc-welders working with their eyes inadequately protected by goggles, and in electricians from short circuit flashes. It is common as snow-blindness in Alpinists and skiers and sometimes follows exposure to mercury vapour lamps.

The symptoms start after a latent interval of some hours. There is intense pain, lachrymation, photophobia and œdema of the lids.

Treatment is simple as the condition is self-limiting. Re-assure the patient that the symptoms will pass off in a matter of hours and that the sight will not be affected. Cover the eyes with cold saline compresses and put the patient to bed. Morphine should be administered if the pain is severe.

CHEMICAL INJURIES

In general, chemical injuries are confined to the cornea and the conjunctiva, particularly of the lower fornix. The cornea is frequently abraded and if the erosion penetrates deeper than the epithelium a permanent scar will result. The conjunctiva is intensely injected and there is usually some discharge. Loss of epithelium in the fornices may result in subsequent symblepharon, a disabling adhesion of the inner side of the lid to the globe.

Treatment.—In any type of chemical injury immediate douching will dilute the irritant and prevent further tissue injury. A tap-water douche at once is better than the theoretical antidote administered with complete asepsis an hour later.

Unless there is actual tissue destruction by strong acids, caustic soda or lime (*see below*) physiological saline is the most effective and least irritating solution to wash out irritants including liquid fuels, spirits, cleaning agents and antiseptics. Certain chemicals which cause actual tissue destruction are with advantage washed out with neutralizing solutions as follows:

For strong acids.—Irrigate freely with sodium bicarbonate 2½ per cent.—*i.e.* half strength Solution of Sodium Bicarbonate B.P. (5 teaspoonfuls to one pint or approximately 4 G. in 500 ml.).

For caustic soda.—Irrigate freely with 5 per cent. ammonium chloride solution.

For lime or plaster-lime.—As this causes severe pain a preliminary instillation of 1 per cent. amethocaine is advisable. All solid particles are first removed using a cotton-wool stick moistened with 10 per cent. neutral ammonium tartrate (the best neutralizing agent). Some of the more adherent particles may have to be scraped off with a spud. The upper lid must be everted and cleaned for quite large plaques frequently adhere there. Finally give a copious irrigation with neutral ammonium tartrate diluted to 1 per cent.

(For eye injuries from spitting snakes *see page 476*, and from indelible pencil *see page 407*).

After removal of the irritant ascertain the extent of the tissue damage by staining with fluorescein (*see page 403*). A corneal abrasion will stain green and a loss of conjunctival epithelium a greenish-yellow. If there is no staining instil a drop of sterile liquid paraffin or castor oil and cover the eye with a lint flap. If



(Photographic Department, London Hospital.)

FIG. 51—Acute conjunctivitis showing the superficial type of congestion most marked in the fornices and brick red in colour.

a corneal abrasion is found insert some Eye Ointment of Atropine 1 per cent. B.P. and apply a pad and bandage. If the conjunctival epithelium in the fornices is damaged symblepharon can be prevented by instilling 1 per cent. amethocaine and gently drawing a glass rod smeared with 6 per cent. sulphacetamide ointment around upper and lower fornix thereby breaking down any adhesions which may be forming. This procedure is repeated every day for a week.

Unless mild it is always safest to refer actual burns for specialist treatment since severe tissue loss in the fornices is best treated by suture of amniotic membrane to the raw surfaces within the first few hours.

INFLAMMATIONS OF THE GLOBE

DIFFERENTIAL DIAGNOSIS OF AN INFLAMED EYE

Four groups of ocular lesions give rise to general congestion of the globe:

- (i) Acute conjunctivitis.
- (ii) Acute keratitis. (The most usual type of keratitis is the single corneal ulcer. The herpetic [dendritic] type often coincides with the onset of a febrile illness. Multiple punctate erosions, a corneal foreign body and the deep or interstitial type of corneal inflammation are also included in the term keratitis).



(Photographic Department, London Hospital.)

FIG. 52—Acute iritis showing the deep or ciliary type of congestion most marked in the peri-corneal zone and purplish in hue. The patient is under treatment with atropine which has caused the pupil to be dilated and irregular.

- (iii) Acute iritis or irido-cyclitis.
- (iv) Acute glaucoma.

These four groups are differentiated by a careful history and examination of *both* eyes (see Fig. 53).

FURTHER DIAGNOSTIC POINTS.

- (i) Discharge. Conjunctivitis is the only one of the four conditions with discharge which varies in amount from a slight crusting of the lids on waking to an intense purulent exudate.
- (ii) Pain. Conjunctivitis gives discomfort only, not pain. The pain of acute glaucoma may be sufficiently severe to cause vomiting.
- (iii) Vision. Vision in glaucoma is greatly reduced. The patient may give a history of coloured haloes around lights.

	Conjunctivitis.	Keratitis.	Iritis.	Glaucoma.
1. Discharge.	Present.	Watery only.	Watery only.	Watery only.
2. Pain.	Discomfort only.	Pain.	Pain + or + +	Pain + + or + + + may cause vomiting.
3. Vision.	Normal.	Depends on area of opacity covering the pupil.	Reduced.	Much reduced.
4. Type of Congestion	Conjunctival.	Ciliary.	Ciliary.	Ciliary.
5. Corneal Transparency.	Cornea normal.	Opacity is localised.	General haze	General clouding.
6. Anterior Chamber	Normal depth	Normal depth.	Normal or deep.	Shallow.
7. Iris and Pupil	Normal size. Normal reactions.	May be small; reacts to light.	Pupil small and often bound. Reflexes poor, or absent.	Pupil dilated. No reaction to light.
8 - Tension.	Normal.	Normal.	May be somewhat increased.	Greatly increased.
9. Examination of other eye.	Condition often bilateral.	May be old scars of previous ulcers.	Pupil may be bound from past iritis.	Disc may be cupped. May be evidence of previous glaucoma operation

FIG. 53

Differential Diagnosis of the Red Eye.

(iv) Type of congestion (see Figs. 51 and 52).

In the conjunctival or superficial type of congestion the redness is maximal at the fornices and the colour a brick red. In the ciliary or deep type the congestion



(Photographic Department, St. Bartholomew's Hospital.)

FIG. 54

Estimation of Intra-ocular Tension.

The patient is directed to look right down. The forefingers are placed on the lid above the tarsal plate. The left forefinger steadies the globe while the right gently palpates. The resistance offered to the palpating finger gives an estimation of the tension.

is circumcorneal in situation and has a violet hue. It is of the utmost importance to recognise this type of congestion (even though in some cases there is a super-added conjunctival congestion) as keratitis, irido-cyclitis and glaucoma are all serious conditions.

(v) Corneal Transparency. Compare the transparency of the two corneæ and note if any opacity is localised

(keratitis) or generalised. Always stain with fluorescein (*pages 43 and 403*) as an active ulcer, especially a small one, can only be seen by this means.

- (vi) Anterior Chamber. Compare the depth on the two sides.
- (vii) Iris and Pupil. There are important differences in iritis and glaucoma. The pupil in iritis is small and the margin may be bound down or seem to be filled with exudate. The pupil in acute glaucoma is dilated, often oval with the long axis vertical. Reaction to light is absent or poor in both cases.
- (viii) Tension (*see Fig. 54*). Compare the resistance of the globe on the two sides.
- (ix) Examination of the other eye. Never omit this, as it often provides important diagnostic clues.

COMMON DIAGNOSTIC DIFFICULTIES.

A small corneal foreign body is easily missed as is an inturning eyelash. Small ulcers, especially those near the corneal margin, are common, and will escape notice unless looked for carefully after staining. Sometimes a conjunctivitis and a keratitis (in the form of a corneal ulcer) may exist together. Further, an acute iritis may give rise to a secondary glaucoma.

EMERGENCY TREATMENT OF THE RED EYE

Congestions, apart from simple conjunctivitis and small ulcers, should receive specialist attention. If this is not available the following measures should be carried out.

Conjunctivitis.—Take a conjunctival swab (for direct smear, culture and sensitivity to antibiotics) before any treatment is started. Cleanse any discharge from the lids with saline swabs. Apply Eye-drops of Penicillin B.P.C. every 3 hours. If the inflammation has not considerably improved in 36 hours the organism is almost certainly penicillin resistant and treatment should be changed to Weak Eye-drops of Sulphacetamide B.P.C. (10 per cent.) four-hourly. Cover with a lint flap. (Never use a pad). [Where there is severe purulent discharge either in the adult or the new born, suspect a gonococcal infection and give the following therapy: Eye-drops of Penicillin B.P.C. every minute for 30 minutes, then every 15 minutes for the next two hours and finally every two hours until the inflammation has subsided. In addition give full doses of sulphadimidine (Sulphamezathine) orally].

Corneal ulcer.—Apply 1 per cent. atropine ointment and Eye-drops of Penicillin B.P.C. three times a day followed by hot steaming, pad and bandage.

Iritis.

Apply 1 per cent. atropine ointment three times a day, followed by hot steamings; cover with a lint flap.

Acute glaucoma.

Instil 1 per cent. eserine in oil into the affected eye two-hourly and 1 per cent. pilocarpine into the opposite eye twice daily since it is also liable to an acute attack. In addition give acetazolamide (Diamox) 500 mg. by mouth and two further doses of 250 mg. at six-hourly intervals. If these measures fail to reduce the tension operation will be necessary. Morphine should be given if pain is severe.

Emergency treatment if diagnosis is in doubt.

Instil Eye-drops of Penicillin B.P.C. four-hourly and give hot steamings. Pad unless discharge is present. Never give atropine if the patient may be suffering from glaucoma, but where this possibility seems unlikely it is quite justifiable to obtain mydriasis with homatropine eye-drops 1 per cent. three times a day.

PAIN IN AND AROUND THE EYE

When eye pain is a presenting symptom the following conditions should be considered.

1. Acute glaucoma (*page 414*).
2. Iritis (*page 414*. Fig. 53).
3. Keratitis (*page 414*).
4. Herpes zoster ophthalmicus (*page 429*).
5. Trigeminal neuralgia.—Here the pain occurs in paroxysms and is sharp or burning and round rather than in the eye. The globe is normal and vision unimpaired.
6. Frontal sinusitis.—Here as well as pain there is tenderness over the affected sinus.

SUDDEN BLINDNESS

(*For Sudden Blindness in Renal Disease see page 271*)

Blindness in one eye.

When of rapid onset this is usually caused by one of the following vascular accidents:

(a) Thrombosis of the central vein.

The disc is swollen, the veins engorged and there are multiple hæmorrhages and exudates along their course. In early cases where the lumen is not completely obstructed anti-coagulant therapy (*see page 595*) has a chance of success.

(b) Thrombosis, spasm or embolism of the central artery.

The disc and retina are pale while the macula shows up as a "cherry spot." The arteries are thread-like. Unless the circulation is restored in 3 hours loss of vision will probably be permanent. Dilatation of vessels may be affected by an inhalation of amyl nitrite or (a measure well worth trying) by massaging the globe with the finger tips through the closed lids and using moderately firm pressure for 10 minutes. Further treatment (in hospital) is by paracentesis of the anterior chamber or by stellate ganglion block (*see page 528*).

(c) Intra-ocular hæmorrhage.

The lesion may be of any size from a small hæmorrhage at the macula to a vitreous full of blood. It may complicate any disease of which bleeding is a feature (*see Chapter IX*).

Conditions causing less sudden blindness in one eye are:

(a) Retro-bulbar neuritis.

Here there may be pain on movement of the globe and a central scotoma. The pupil reacts sluggishly to light, but the consensual reaction is normal. Treatment is that of the causal condition

(b) Detachment of the retina.

This is characterised by progressive loss of the visual field and needs immediate treatment in hospital for reattachment.

(c) Central choroiditis.

A patch of choroiditis near the macula or disc may cause a loss of vision which increases over a period of days.

(d) Hysteria.

Hysterical blindness may be partial or complete and is occasionally bilateral. The patient avoids obstacles in his path and shows the blink reaction to menace. The pupils and fundi are normal.

Blindness affecting both eyes.

This is almost always due to general causes such as:

Toxic substances—methyl alcohol (*see page 12*), quinine, and (rarely) lead, arsenic, carbon disulphide, ergot, and filix mas.

Severe hæmorrhage particularly from the gastro-intestinal tract (*see page 92*), uterus and lungs. The blindness comes on several days after the main loss.

Hypertensive states and toxæmia of pregnancy.

Renal disease (*see page 271*).

Diabetes.

Diseases of the central nervous system such as acute encephalomyelitis (*see page 198*) and (more insidiously) Leber's disease and Devic's disease.

J H. DOBREE.

CHAPTER XXIII

Emergencies in Skin Disease

SKIN diseases are rarely fatal in themselves but they may be of urgent importance because

1. Pathological conditions of viscera may coexist, *e.g.*, in lupus erythematosus, the reticulosos, epitheliomata and melanomata, and in toxic and allergic dermatoses.
2. Secondary infections may develop, *e.g.*, from tinca pedis or pompholyx.
3. Loss of fluid, electrolytes and protein may complicate extensive moist eruptions such as pemphigus.
4. The skin eruption may give the patient much anxiety or it may be the outward evidence of an emotional disturbance.
5. It is sometimes an urgent matter to differentiate a non-infectious skin disease from an infectious exanthem.

THE DIAGNOSIS OF ACUTE ERUPTIONS

A discussion on the finer points of differential diagnosis cannot be made in a book of this nature ; but a list of the more likely causes for various types of eruption may prove of value in emergency. An eruption for which immediate treatment is expected may be widespread (one of the specific fevers, a toxic erythema, a specific skin disease or a generalised eczema) or it may be local (of bacterial or fungous origin, or eczematous in nature).

WIDESPREAD ERUPTIONS

Erythematous.—Scarlatina ; morbilli ; rubella ; and toxic eruptions from drugs or infections which may simulate these diseases or which may present as urticaria, purpura, erythema multiforme or erythema nodosum ; the roseolar syphilide.

Scaly erythematous.—Pityriasis rosca and its mimicry by drug eruptions and syphilides ; seborrhœic dermatitis ; guttate psoriasis ; peeling scarlatina and lichen planus.

Papulo-vesicular.—Widespread eczemas from internal or external causes, including especially eruptions secondary to hypostatic

eczema (eczematides) or arising from the prolonged or widespread use of sensitizing or occlusive applications; dermatophytides; varicella; variola; papular urticaria; halogen eruptions; syphilis; also the rare condition of pityriasis lichenoides et varioliformis acuta.

Bullous.—Insect bites; urticaria; severe erythema multiforme (Stevens-Johnson syndrome); varicella; variola; vaccinia; Kaposi's varicelliform eruption (generalized herpes); drug eruptions; plant dermatitis; and juvenile dermatitis herpetiformis.

LOCALIZED ERUPTIONS

Infective dermatoses are, as a rule, crusted or exudative and patchy in distribution, with peripheral "satellites." Eczematous dermatoses are usually papulo-vesicular or erythematous-squamous, and are more diffuse and ill-defined, with the exception of discoid eczema of the extremities in which sensitization to skin organisms seems to play a part. Mixed eczematous and infective conditions are not uncommon. With solitary or ringed lesions, likely diagnoses include tinea, impetigo, pityriasis rosea, syphilis, psoriasis, lichen planus, erythema multiforme, lupus erythematosus, and granuloma annulare. The differential diagnosis of these eruptions depends on the history, and on the distribution and character of the lesions. In the diagnosis of fungous infections physical signs alone cannot be relied upon and it is advisable to take a scraping and examine the specimen in liquor potassæ microscopically, using $\frac{2}{3}$ and $\frac{1}{10}$ objectives. A fixed or recurrent localized erythema may be caused by phenolphthalein, iodides, barbiturates or sulphonamides.

DRUG ERUPTIONS

Drug eruptions are usually mild and transient but may be severe and even fatal because a vital organ or tissue is also affected, particularly the blood-forming tissues, vessel walls, kidneys, liver, or the heat-regulating centre. In particular, this applies to amidopyrine, antibiotics, arsenic, barbiturates, carbromal, gold, halogens, mepacrine, *para*-aminosalicylic acid, phenolphthalein, phenylbutazone, quinine, and sulphonamides. The list continually changes with the introduction of new drugs and the abandonment of old ones.

Arsenic and the halogens cause characteristic eruptions, but with most other drugs the eruptions are non-specific and to identify the

cause reliance must be placed on the history, perhaps followed by a sub-therapeutic ($1/10$ th) test dose at a later date.

A possible drug causation should be considered in erythematous eruptions, whether morbilliform, scarlatinaform, urticarial, purpuric, or erythema multiforme-like; in stomatitis; in scaly eruptions resembling pityriasis rosea, seborrhœa, psoriasis, or lichen planus; in exfoliative dermatitis, eczematoid, furuncular, acneiform or rosacea-like conditions, and with granulomatous lesions. Herpes simplex and zoster may be precipitated by drugs; and acné vulgaris, rosacea and seborrhœic dermatitis may be aggravated by them. Pruritus may be caused by opiates, including codeine.

Before giving treatment, an attempt should be made to decide whether a drug eruption is due to (1) direct toxic or allergenic action on the skin, or (2) the indirect effects on the skin of toxic or allergenic properties of the drug on other organs.

Direct toxic action on the epidermis may cause exfoliative, dyskeratotic and pigmentary changes. Allergic hypersensitivity causes eczematous changes in the epidermis and erythema, urticaria or purpura in the dermis.

Toxic action on the blood-forming tissues may cause purpura and cutaneous or oral infections, or ulcers. On the liver it may cause seborrhœic, exfoliative, lichenoid and phrynodermatous eruptions, photosensitivity, cheilitis, angular stomatitis and anogenital pruritus. Oedema and hæmaturia may result from renal involvement. Myxœdema may appear if the thyroid gland is damaged and unexplained pyrexia may occur if the heat-regulating centre is involved. Antibiotics, especially the tetracyclines and chloramphenicol, may so disturb the bacterial flora of the bowel as to allow monilia to proliferate and cause oral moniliasis or anogenital pruritus. A pellagroid state sometimes develops.

Treatment.—In severe cases the patient should be confined to bed and given ample fluids and a light diet. All suspected drugs should be withheld. Local treatment consists of bland, non-sensitizing applications. The specific antidote should be used if there is one, for example, Dimercaprol (B.A.L.) (*see page 265*). Antihistamine drugs (*page 611*) are helpful for urticarial and erythema multiforme-like eruptions. Crude liver injections, 4 ml. daily, and vitamin supplements by mouth are indicated when a drug-induced nutritional deficiency is likely. Blood transfusion and

vitamins C and K (*pages 656 and 657*) may be helpful if there is evidence of damage to the bone marrow (*see page 176*). Cortisone is sometimes indicated, *e.g.* in gold dermatitis.

THE RELIEF OF ITCHING

Keep the patient at rest in cool surroundings. A cradle over the legs may be helpful, and nightwear of silk or cotton should be worn. If the itching is of epidermal origin (acute vesicular eczema), wet dressings and lotions are best, such as physiological saline; lead and spirit lotion, B.P.C.; a calamine lotion, without phenol; or oily calamine lotion, B.P.C. A trustworthy formula is :—

Prepared calamine	2 G. (gr. 30)
Lime water	14 ml. (m 240)
Olive oil	to 30 ml. (1 fl. oz.)

Hydrocortisone lotion 0.5 to 1.0 per cent. is most valuable in acute eczematous dermatitis as a suppressant of the excessive inflammatory process and for relief of the smarting and itching.

Adequate sedation should be given with phenobarbitone 32 to 65 mg. (gr. $\frac{1}{2}$ to 1), or promethazine 10 to 25 mg. twice a day, and butobarbitone (Soneryl) 0.2 G. (gr. 3) in the evening.

In itching of dermal or of combined dermal and epidermal origin (urticaria, erythema multiforme, prurigo), calamine lotion B.P. is useful. Hydrocortisone ointment may relieve prurigo but is ineffective in urticaria and in erythema multiforme. Internally, antihistamine drugs (*page 611*) should be given in a dosage just sufficient to control the symptoms, with gradual reduction as these are relieved. In pruritus without obvious skin abnormality relief mainly depends on adequate sedation by mouth but the patient may like to apply a lotion such as lead and spirit lotion B.P.C.

SOME CONTRA-INDICATIONS IN THE TREATMENT OF ACUTE SKIN CONDITIONS

Local applications.

(i) Ointments should not be used in acute cases. They prevent evaporation and so increase itching and encourage or spread infection. Lotions or powders should be used in acute cases and emulsions or creams in subacute cases.

(ii) Antiseptic or antibiotic applications should not be used unless infection is clearly present. In infected eczema Vioform cream (Ciba) is often helpful. Antibiotics should only be used topically under bacteriological guidance. While awaiting this information, a quaternary ammonium compound (for example, cetrimide 1·0 per cent.) can be used, and oily calamine lotion subsequently applied.

(iii) It is wise to avoid using sulphonamides, flavine, mercurials and penicillin topically because of the risk of sensitization.

(iv) Benzocaine antipruritic applications are very liable to sensitize the skin. The topical use of antihistamine drugs is also fraught with this risk.

(v) The over-treatment of tinea pedis with powerful fungicides may cause a generalized eruption. If there is secondary infection, soaks of potassium permanganate 1 in 5,000 (gr. 2, or 3 or 4 crystals per pint) are useful; or if there is no infection, the application of magenta paint at bedtime and 3·0 per cent. salicylic acid powder in the morning is often effective.

(vi) Emulsifying ointment is the best base for use in hairy areas, where shake lotions or pastes cause unpleasant matting and are difficult to remove.

(vii) The use of "blunderbuss" prescriptions should be avoided. They increase the risk of sensitization and if this occurs it is difficult to find which ingredient is responsible.

(viii) Lead and sulphur should not be used in the same lotion because a black deposit of lead sulphide results.

(ix) Mercurials or dithranol should not be used in subacute psoriasis as exfoliative dermatitis may follow.

A few examples of eczematization commonly resulting from faulty and perhaps over-vigorous treatment may be helpful :—

Herpes febrilis may become impetiginized, but if uncomplicated herpes is misinterpreted as impetigo and treated with an antibiotic a rapid local spread of eczema may follow on the face and even on the limbs.

The herald spot of pityriasis rosea is sometimes diagnosed as ringworm and treated with a fungicide. If the eruption that follows is interpreted as a spread of the infection and similarly treated, an obstinate eczema may be superimposed on this relatively trivial, self-limited skin disorder.

Hypostatic ulcers are often treated by patients and others with antiseptic applications, following which there is a localized spread of eczema which may suddenly become generalized in explosive fashion. This particularly occurs after the application of sensitizing drugs or occlusive dressings to extensive moist surfaces.

Systemic treatment.

(i) Unless their use is essential, sulphonamides should not be used systemically if sensitization has occurred from their previous topical application.

(ii) Drugs (except antihistamines) should not be given in acute urticaria and other acute eruptions suspected of being toxic in origin.

(iii) Sufferers from acute dermatoses should be protected from sunlight, heat, humidity, exertion and excitement.

PRICKLY HEAT (Miliaria Rubra)

This disorder is common in the tropics and is sometimes seen in temperate climates as a complication of dermatitis. The onset may be sudden. Relief may be obtained and sweating occur following the inunction of lanolin, ointment of wool alcohols, or hydrous ointment. The underclothing should be light and pervious; excessive washing should be avoided, and lotions containing spirit, antiseptics and powders should not be used.

CHILBLAINS (Erythema Pernio)

Chilblains are caused by cold and damp acting on those predisposed to them by poor peripheral circulation. As a first-aid remedy the affected parts should be cleaned and massaged gently from the periphery with olive oil or oily calamine lotion B.P.C.

ACUTE CONTACT DERMATITIS

From a carefully taken history and the distribution of the eruption, it may be possible to determine the cause and prevent further exposure. Acute dermatitis of the scalp and neck may be caused by the dye paraphenylenediamine used on the hair or in furs. It may also arise from permanent-wave solutions. Acute dermatitis mainly involving the face and neck may be due to airborne irritants, including some plants (most commonly, the primula and chrysanthemum), occupational and other dusts, formalin vapour, and insecticides. It may also arise from hand-transferred

substances including nail varnish, hair lacquer, and other cosmetics, and many substances handled in work and hobbies, including alkalis, glues, photographic chemicals, and procaine. Dermatitis around the eyes may result from the use of eye lotions containing sulphonamides, antibiotics, silver, mercury or atropine. Nurses sensitive to streptomycin usually present with conjunctivitis or dermatitis of the eyelids. Neurodermatitis also occurs on the eyelids, closely mimicking contact dermatitis. Acute dermatitis of the face and other exposed parts, with light sensitivity, may result from the topical use of sulphonamides, promethazine, mercury or flavine. Acute erythema of the face may also follow emotional crises without obvious contact cause. Dermatitis of the hands and forearms arises from innumerable occupational irritants, but rarer causes such as plant dermatitis and the handling of nickel-plated, chromium-plated and rubber objects must not be forgotten. Acute dermatitis elsewhere on the body may follow the use or abuse of antiseptics and parasitocides in soaps, lotions and ointments, and counter-irritants, especially mercurial ointments, mustard plasters, turpentine liniments, and camphorated oil.

Treatment.—After removing the cause, one of the lotions (*see page 423*) should be applied. For cleaning the skin, olive oil should replace soap and water during the acute phase. When the condition is resolving, a non-scented superfatted soap should be used with tepid water. The patient should rest and avoid exposure to sunlight, cold winds, or the warmth of fires.

A 0.5 or 1.0 per cent. hydrocortisone ointment or lotion is a valuable application for acute eczematous dermatitis but not for application to skin damaged by a primary irritant, for example, an alkali. If secondary infection is present, the appropriate antibiotic should be applied according to the bacteriological findings. It is advantageous in infected eczematous conditions to mix hydrocortisone with the antibiotic.

Adequate sedation is usually obtained with phenobarbitone 32 to 65 mg. (gr. $\frac{1}{2}$ to 1) twice or three times a day, and butobarbitone (Soneryl) 0.2 G. (gr. 3) at night.

ACUTE WIDESPREAD SENSITIZATION DERMATITIS

This is most commonly seen after the use of sulphonamides, flavine, picric acid, benzocaine, antibiotics, antihistamines, or ammoniated mercury on the skin, or from dyeing of the hair with

paraphenylenediamine. Generalized eczema may also originate from secondarily infected or over-treated hypostatic ulcers and eczema, or from the use of occlusive dressings, *e.g.*, paraffin ointment (autolytic eczema).

The same local principles apply as for treatment of acute contact dermatitis. Hydrocortisone lotion should be applied to the worst areas, if the extent of the eruption makes it economically impractical to apply it to all the lesions. A daily intramuscular injection of crude liver extract, 2 to 4 ml. for about 7 days, may hasten resolution. Sedatives should be prescribed.

ACUTE URTICARIA AND ANGIONEUROTIC OEDEMA

Acute urticaria most commonly arises from gastrointestinal allergens—food (especially shell-fish, strawberries, wheat, eggs, milk, chocolate), drugs, or intestinal parasites; from injected allergens—drugs and sera, or from bites. Inhaled allergens sometimes cause it. The cause may be obvious and easily removed but an emotional upset can also be solely responsible. If a gastrointestinal cause is suspected, a dose of 30 ml. (1 fl. oz.) of White Mixture, B.P.C., should be prescribed. The patient should be kept at rest, preferably in bed, on a bland diet, and an antihistaminic drug prescribed in the dose that is found to control the lesions (*see page 611*). Any other drugs which the patient is taking by mouth, injection or inhalation should be stopped.

Locally, lead and spirit lotion, B.P.C. (*see page 424*) or calamine lotion, B.P., are suitable. If the lesions are very extensive, or involve the mouth, tongue or throat, 0.1 to 0.5 ml. (2 to 8 minims) of Injection of Adrenaline B.P. should be given subcutaneously, in a single dose; or by repeated injections of 0.06 ml. (1 minim) every minute until the lesions are controlled.

ACUTE INFECTIONS

Infected pompholyx and infected epidermophytosis.

Vesicular eruptions about the hands and feet often become secondarily infected, leading to local pyoderma, lymphangitis, lymphadenitis, abscess or cellulitis (erysipelas). The patient should rest, with the affected part elevated. If the infection is superficial, purulent bullæ should be incised and drained, and the affected part soaked for 10 minutes twice a day in a solution of potassium permanganate 1 in 5,000 (gr. 2, or 3 or 4 crystals per pint; or

enough to make a pink but not red solution), and then dressed with the most suitable antibiotic, depending on the bacteriological findings. In the presence of lymphangitis and lymphadenitis, the arm should be kept in a sling or the affected leg supported on pillows, and the patient should receive either intramuscular injections of procaine benzyl penicillin 300,000 units daily until the condition has subsided, or a course of sulphadimidine (Sulphamezathine) (*see page 599*).

Boils on the nose and lip.

Boils about the nose need special care, owing to the danger of spread through the superficial and deep venous anastomoses. All boils in this area should be treated with the utmost respect and the sufferer kept in bed and strongly warned against the risk of manipulation. Pledglets of wool soaked in hot saline may be gently applied. The area should be immobilized as far as possible, talking minimized, and the patient fed with liquids. All general measures must be employed to encourage resolution, including penicillin, tetracycline, or some other suitable antibiotic (*see page 601*). When the boil starts to discharge, frequent applications of warm hypertonic saline should be made without squeezing or pressure.

Cutaneous diphtheria.

Cutaneous diphtheria is most commonly a complication of nasal diphtheria or of a nasal carrier state, though it may occur by itself. The lesions may be ulcerative, impetiginous, or eczematoid. There is often a blood-stained nasal discharge. Culture and animal virulence tests are necessary but treatment (*see page 309*) should be started on clinical evidence alone. Locally, the most suitable antibiotic should be applied to clear the associated coccal infection. Three or more consecutive negative skin and nasal swabs should be obtained before the patient is discharged.

Erysipelas.

In every case, search should be made for fissures in the vicinity. They should be treated with a 2 per cent. solution of silver nitrate in spirit of nitrous ether. Erysipelas with constitutional disturbance necessitates rest in bed, warmth, fluids, and a course of penicillin or sulphadimidine (Sulphamezathine) (*see page 599*).

Erysipeloid.

This occupational disease of those who handle or prepare food is caused by the introduction through a prick or abrasion on the

hand of the organisms of swine erysipelas. The spreading redness, swelling and discomfort may be mistaken for erysipelas or for a purulent infection of the finger. The condition responds rapidly to intramuscular injections of procaine benzylpenicillin 300,000 units daily for up to a week.

Herpes Zoster Ophthalmicus.

Herpes zoster affecting the first branch of the trigeminal nerve is often of the severe gangrenous type and there is a risk of involvement of the cornea, possibly leading to destruction of the eye. The patient should be nursed in bed and the eye examined for evidence of corneal involvement, this being more likely to occur in cases with lesions on the nose.

Should the eye be involved, 2 per cent. homatropine drops should be instilled and the assistance of an ophthalmic surgeon obtained.

The sloughs take several days to separate and every care must be taken to prevent any superimposed infection or retention of discharges under occlusive dressings. For this reason, wet dressings, such as slightly hypertonic saline or freshly prepared potassium permanganate 1 in 5,000 (*see page 429*) are best. For the non-gangrenous areas, calamine lotion, B.P., is suitable. Collodion should only be used on unbroken lesions in mild cases. When the sloughs have separated, a sterile yellow soft paraffin 2 mm. mesh tulle gras dressing may be found most comfortable. Antibiotic applications are useful for controlling secondary infection but, whether administered topically or orally, they have no beneficial effect on the zoster itself.

The following drugs (amongst others) have the reputation of relieving the severe pain and of shortening the course of zoster, if they are administered intramuscularly at an early stage for three or four days: Pituitary (posterior lobe) B.P.C., 10 units; vasopressin, B.P., 5 to 15 units; crude liver extract, 4 ml.; or cyanocobalamin 50 micrograms. For the relief of pain, codeine tablets may be adequate, or the following mixture, every six hours:

Soluble Phenobarbitone	32 mg. (gr. $\frac{1}{2}$)
Phenazone	0.26 G. (gr. 4)
Tincture of Gelsemium	0.6 ml. (10 m)
Syrup of Lemon	2 G. (gr. 30)
Peppermint Water	to 15 ml. ($\frac{1}{2}$ fl. oz.)

Chlorpromazine, 25 mg. t.d.s., may make the pain more bearable, but it is sometimes necessary to give heroin (diamorphine) 11 mg. (gr. $\frac{1}{6}$) sublingually, or even morphine parenterally, in order to obtain a night's rest.

SPECIAL HAZARDS OF THE ECZEMA-PRURIGO PATIENT

Infants suffering from eczema are especially susceptible to skin infections and sudden death may occur, apparently from overwhelming general infection, the skin being the portal of entry. After exposure to herpes simplex an eczematous infant may develop Kaposi's varicelliform eruption—a generalised form of herpes with a considerable death rate. Vaccination of an eczematous child may be followed by generalised vaccinia.

Because of these risks, it is inadvisable to admit to hospital children who are suffering from eczema, particularly if under one year of age. The contact of eczematous children with recently vaccinated individuals or with those suffering from herpes simplex, zoster or varicella, should be avoided. Vaccination of such children should be deferred until the skin has been clear for at least a year. Any superimposed infection in an eczematous child should be treated topically with the appropriate antibiotic. Cubicle nursing is desirable. Kaposi's varicelliform eruption and eczema vaccinatum call for a high standard of nursing care and suitable antibiotics to combat secondary coccal infection.

URGENT SKIN CONDITIONS ASSOCIATED WITH PREGNANCY

Herpes Gestationis.

Herpes gestationis usually appears in the third trimester but it may start earlier or may only develop in the puerperium. It is an intensely itchy, polycyclic, erythematous eruption, with bullæ. The distal parts of the limbs may be especially affected, or the eruption may be widespread on the trunk and limbs.

Cortisone should be given in a dose sufficient to give partial relief: up to 150 mg. a day may be necessary. It is advisable to tail off this dosage before delivery. Good results have also been reported from Progesterone up to 100 mg. daily, later reduced to a maintenance dose of between 25 to 50 mg. daily. The patient should not take iodides or bromides in any form. Oestrogens may cause an exacerbation. Local treatment consists in the application of Lotio Calaminæ (N.F.).

Impetigo Herpetiformis (Hebra).

This is a very rare condition usually occurring with pregnancy. Irregular polycyclic clusters of small pustules are present mostly in the flexures, each lesion being surrounded by an inflammatory halo. Ulcers may appear in the mouth. The lesions are sterile. There is grave constitutional disturbance with fever, vomiting, diarrhoea, nephritis and splenomegaly. Hypoparathyroidism is often present. The malady is sometimes fatal.

Calcium in large doses and dihydrotachysterol (A.T.10) 5 to 20 drops daily are said to help the condition. Antibiotics should be used topically for any secondary infection and a trial of tetracycline by mouth would seem justifiable. If the disease occurs in the first trimester it may be advisable to terminate pregnancy. If it develops later, this procedure may be too hazardous.

BRIAN RUSSELL.

BURNS

The emergency treatment of burns consists in the prevention of shock, the prevention of infection and further damage by mechanical and chemical trauma, and the replacement of areas of full thickness skin destruction at the earliest moment.

Shock.—This is the commonest cause of death in large burns and the first complication to be prevented. For first-aid treatment the extensively burned patient should be wrapped in a clean sheet, kept warm with blankets if necessary, and taken to hospital immediately. The supreme need is early and adequate transfusion, and local treatment takes second place. One effective dose of morphine may be given intravenously if the patient complains of pain, and oral fluids may be given to relieve thirst but to discourage vomiting should be limited to three ounces an hour for children and five ounces an hour for adults.

The treatment of patients who will become shocked if not given intravenous therapy is made easier by the rough relationship which exists between the area burned and the amount of plasma lost. A child with more than 10 per cent. of the body surface burned, or an adult with more than 15 per cent., excluding erythema, will require early transfusion with plasma or dextran, and possibly blood, if clinical shock is to be prevented. The estimation of these areas is made easier by using the "Rule of Nines" method (Fig. 55) and by using for measurement the area of the patient's outstretched hand, which is about 1 per cent. of his body surface.

Transfusion should start at once and must continue as long as there is appreciable loss of plasma, which is usually 24 to 36 hours. The rate may be controlled by repeated hæmatocrit readings using peripheral capillary blood from any area of warm skin. A surface

area formula may help, *e.g.*, in *adults* a total of 1 to $1\frac{1}{2}$ litres of colloid is usually needed for each 10 per cent. of the body surface burned. Furthermore, certain clinical signs are of value; for example, pallor, thirst, a cold nose and forehead, and restlessness often signify inadequate colloid replacement. Oliguria is commonly found but if the urine output is over 30 ml. per hour fluid administration is probably adequate. In practice a combination of these guides is usual.

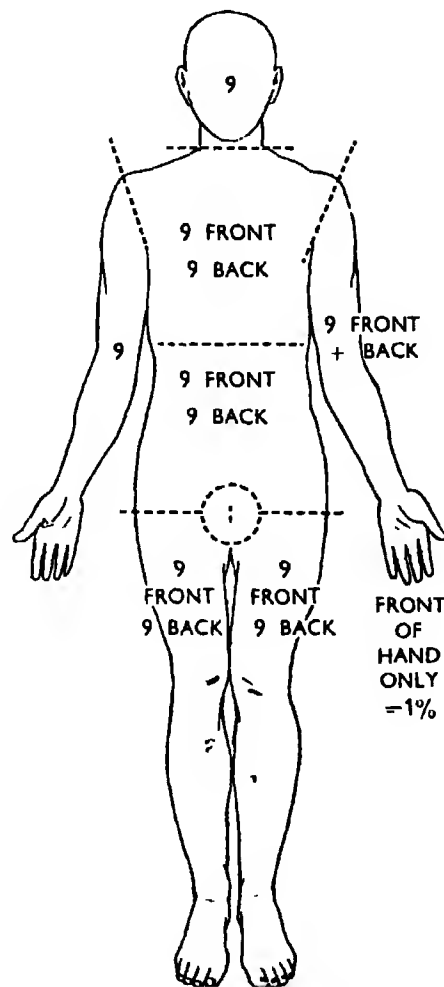


FIG. 55
The Rule of Nines.

Infection.—Ninety per cent. of recently burned patients arriving at a Burns Unit had no pathogenic organisms on the burned surface. In other words, *a new burn is a clean wound*, but one which is especially liable to infection because of its large area. Bacterial contamination may result from direct contact with hands and clothes, or by airborne spread from mouths and noses.

To prevent this complication, first-aid treatment should afford immediate covering of the burn with a sterile or freshly laundered cloth until the lesion can be examined for the diagnosis of its depth, and then properly dressed or grafted.

There are at present two principal local treatments, the "closed" and the "exposure" methods. Choice should be made between these two by considering which method can be carried out most efficiently for the particular burn, patient, and environment. The "closed" method aims at covering a clean wound with an efficient antibacterial barrier of penicillin cream, gauze, dry cotton wool, and crepe bandage and, providing this dressing remains efficient, it is changed as infrequently as possible. The "exposure" method is based on the principle that since it is difficult to prevent bacterial colonisation completely with a dressing, an alternative is to secure drying of the burned surface by exposure. Bacterial growth will then be limited by the dry and cool environment and exposure to light. Success with the "closed" method depends on sufficient dry wool to prevent the exudate soaking through; with the "exposure" method it depends on adequate immobilisation to prevent cracking of the scab. The "closed" method is of universal application except for burns of the face, buttocks and genitalia, and in hot climates; the "exposure" method is an in-patient treatment especially valuable where closed dressings are difficult and for burns of one side of the trunk only. Neither method requires an anæsthetic.

Since the presence of dead tissue encourages the growth of organisms, whenever possible a deep burn should be excised and grafted at once as the best method of preventing infection.

Skin grafting.—The modern classification of burns is into two categories, partial-thickness skin loss which heals without scarring from remaining epithelial elements in the burned skin, and full-thickness skin loss which heals slowly by contraction of the wound and epithelial cell migration from the edge.

Full-thickness burns should be grafted as soon as possible to limit infection, fibrosis and scarring, and this can always be started by the end of the third week after removing the granulation tissue.

In about a quarter of these deep burns, however, it is possible to shorten the healing time by excision of the burn on the day of injury, and immediate closure of the wound by skin grafting. This procedure can be carried out on burns up to 15 per cent. of the

body surface, provided one can be certain that the depth of burning is full-thickness skin loss. This emergency treatment gives the best result in the shortest time, and is one of the few important recent advances in the treatment of burns.

Chemical burns.—Immediate vigorous washing with water is the emergency treatment for caustic burns. This should be followed by a compress of buffer phosphate solution for 20 minutes. This solution is non-irritant and can be used in the eyes. (Composition: KH_2PO_4 , 70 G.; $\text{Na}_2\text{HPO}_4 \cdot 12\text{H}_2\text{O}$, 180 G.; in 850 ml. of water: this makes 1,000 ml. of solution). Further treatment is the same as for thermal burns. For hydrofluoric acid burns *see page 358*.

D. MACG. JACKSON.

CHAPTER XXIV

Emergencies during Anaesthesia

THE modern anaesthetist carries a large share of responsibility for the patient's life during an operation. He should, therefore, anticipate every danger and also learn to avoid emergencies by a wise choice of the anaesthetic he uses. Emphasis is placed on this aspect in the pages which follow.

RESPIRATORY OBSTRUCTION

Inflammatory swellings.

Pressure of inflammatory swellings (*e.g.*, Ludwig's Angina) on the upper respiratory tract may precipitate a dangerous situation during the induction of anaesthesia. If there is any suggestion of dyspnoea, local anaesthesia is the only safe method and in any case a tracheotomy set should be ready. The choice of anaesthetic is difficult; intravenous anaesthesia with or without curare will almost certainly kill the patient at once. Chloroform by the open method is fairly satisfactory. The delivery of oxygen under the mask is an advantage. Whatever method is used, an essential preliminary is to arrange the patient's position on the table so that he can breathe most easily.

Intra-pharyngeal inflammatory swellings may cause trismus from spasm of the masseter muscles. The patient should be shown how to use a boxwood wedge mouth opener himself. When he has opened his mouth sufficiently, a gag can be inserted and the abscess drained under surface anaesthesia.

Fractured mandible and oral lacerations.

Injuries to the upper parts of the respiratory tract may suffocate the patient once his protective reflexes are diminished by anaesthesia. Two essential preliminaries are to make up blood loss by transfusion and to empty the stomach and leave a wide bore tube in the oesophagus.

The head of the table is then lowered and injections of thiopentone and suxamethonium allow intubation with a cuffed tube, the initial dose of thiopentone being given fairly quickly. If the patient's general condition is poor the speed and the dose must

be reduced. It may be necessary for an assistant to hold a laryngoscope partly in the pharynx with continuous suction to keep a clear field during the injection. As soon as the tube is passed and the cuff inflated, endotracheal suction is applied to aspirate any fluids inhaled at the time of injury. After suction, artificial respiration by intermittent bag compressions must be kept up till respirations return. On no account should the throat be sprayed by a local anæsthetic; if the dosage of anæsthetic agents is kept to a minimum, the protective reflexes are more likely to return rapidly after operation.

Asthma.

As a general rule surgery can wait until after an attack of asthma has responded to treatment (*see page 127*). Between attacks the subject tolerates anæsthetics normally, although a paroxysm may be set up. Procaine (1 G. in 560 ml. (1 pint) of physiological saline) by intravenous drip is used to prevent or treat this type of spasm during anæsthesia. Adrenaline (*see page 127*) may be of great value but should not be given in the presence of chloroform, trichlorethylene, or cyclopropane, and the same probably applies to aminophylline (*see page 128*). Parry Brown gives all known asthmatics thiopentone after the injection of 1 ml. of 10 per cent. cocaine through the cricothyroid membrane, and maintains anæsthesia with nitrous oxide oxygen and ether with a relaxant. He has used cocaine effectively to relieve the spasm induced by bronchoscopy in patients under thiopentone anæsthesia with suxamethonium.

Rarely is anæsthesia needed during an attack of asthma; it is a hazardous undertaking. Local anæsthesia is first choice, otherwise if good facilities do not exist, open ether with nasal oxygen is fairly satisfactory but difficult. Premedication by morphine is contra-indicated; atropine is essential, and if speed is necessary it can be given intravenously.

Helium, measured by the nitrous oxide rotameter (multiply the readings by 3.3), with oxygen and ether using Boyle's apparatus reduces the work of breathing and is preferable to cyclopropane which increases bronchospasm. Relaxant drugs may liberate histamine and are potentially dangerous.

Tracheal collapse and recurrent laryngeal nerve injury.

A patient who is markedly dyspnoeic immediately after removal of an endotracheal tube following partial thyroidectomy is

likely to have either tracheal collapse, or damage to both recurrent laryngeal nerves. The tube should be replaced immediately. If the vocal cords are seen to be in the cadaveric position, tracheotomy will have to be carried out; if not, the tracheal rings have collapsed and must be sutured at once to the overlying structures in the neck.

Endotracheal obstruction.

Endotracheal anaesthesia requires special skill. A perfect airway is not guaranteed by intubation, for the tube is a fixed passage and does not expand like the trachea with increased respiratory activity; also its walls offer a slight obstruction which increases when the surrounding cuff is inflated.

Intubation by laryngoscopy without injury needs a certain degree of relaxation of the jaw muscles and the laryngeal reflexes must not be active. With thiopentone alone, the required dose may be dangerous, but if a small dose is used, the patient is liable to clamp the tube in his jaws. Then asphyxial spasm follows rapidly and if a relaxant cannot be injected intravenously, the mouth must be opened with a gag, the tube removed and oxygen given by a mask. A similar disaster may overtake a patient who has been sent back to the ward with a tube in his mouth. Death has occurred after operation because a tube has been inadvertently left inside the patient.

Tubes must be selected with care because after repeated boilings they tend to bend in the pharynx! If re-intubation is not practicable it may be possible to slide a wire coil over the proximal part of the tube as recommended by Bourne, or to extend the head and flex the neck to bring the upper respiratory passages into a straight line.

Assistants who lean on the covering towels may bend a tube issuing from the nose or mouth, unless the strapping is arranged so that only the metal angle piece at the top of the tube projects.

An over-inflated cuff may bulge over the distal opening or it may injure the tracheal mucous membrane causing necrosis. Subsequently the oedema may necessitate tracheotomy at a lower site. The cuff may burst and to this accident one death has been attributed. Inflation of the cuff by gradual small increments of air from a syringe as oxygen is applied by positive pressure from a bag, till it no longer escapes with each positive phase, should avoid over-inflation.

A long endotracheal tube passed into one bronchus may obstruct the other, causing collapse of a lobe of the lung. Tubes supplied by the makers must be shortened before use, gauging the correct length by placing the tube at the side of the neck. If laboured breathing develops on intubation, the tube should be withdrawn slowly, and, as the distal end passes the carina, the sounds will at once increase as the bronchus opens.

Moist respiratory sounds mean fluid in the tube which must be sucked out at once; in infants the tubes are so small that the fluid has to be removed through a channel the size of a ureteral catheter. Before extubation the pharynx should always be sucked dry.

Obstruction developing during anæsthesia.

Prompt action is often required to relieve respiratory obstruction during anæsthesia. The actual obstruction, such as approximation of the tongue and posterior pharyngeal wall or approximation of the vocal cords, is rarely seen during a routine administration, though it can be readily demonstrated by laryngoscopy. Lack of attention may allow the condition to continue, although there may be some difficulty in determining what is wrong. Any suspicion that obstruction is due to a foreign body calls for immediate laryngoscopy and, if necessary, bronchoscopy with postural drainage in the case of inhaled pus, blood or vomit.

DIAGNOSIS OF OBSTRUCTION AND ITS SITE.—Obstruction during anæsthesia is recognised by changes in respiration. The quantity of respired gases passed exteriorly becomes reduced simultaneously with an increase in the force of respiratory movements, though this is sometimes difficult to demonstrate when respiratory depressants like thiopentone and cyclopropane are used. Normal free movements are replaced by slight straining movements. The belly component of respiration increases at the expense of the chest part, producing what is known as "reversed" respiration.

Alterations in the quantities of respired gases can be roughly assessed by observing changes in the excursions of reservoir bags, or by noting the change in the rush of air in the case of open methods. When obstruction is complete, respiratory movements may continue for a time with a variable force depending on the depth of anæsthesia and the agent used, but no gases pass and no sound can be heard.

The site of obstruction indicates the remedial measure and can often be predicted by abnormal respiratory sounds. Partial obstruction in the pharynx produces a harsh sound known as stertor, which is devoid of any vocal element. Turning the head to one side allows the base of the tongue to fall to the lower part of the pharynx, and pushing or pulling the jaw forward carries the tongue away from the pharyngeal wall, with the disappearance of stertor. Asphyxia results in spasmodic contractions of many muscles, including those which constrict the respiratory passages. I have seen the base of the tongue pulled taut to the soft palate in asphyxial spasm. This may be one explanation of the failure of simple postural changes in clearing the passage. The purpose of a mechanical pharyngeal airway is to separate the approximating parts, but if the instrument is too short, it is easy to see how the dorsum of the tongue could slide over the distal end and still obstruct, and how a large airway may push the epiglottis back over the glottic aperture. Enough has been said to warn the reader that the insertion of a mechanical pharyngeal airway does not always solve the problem of pharyngeal obstruction. The Waters' type of airway (Fig. 56) is the simplest to slide over the dorsum of the tongue and requires the least separation of the teeth. Masseteric spasm may prevent the insertion of a pharyngeal airway. A well lubricated cut Magill tube passed down one nostril will get over the difficulty. It may evoke a fit of coughing and in that case it should be withdrawn slightly and the depth of anaesthesia rapidly increased. Sometimes a harsh sound having a nasal element clears up after the passage of a cut Magill tube, thus indicating an obstruction by the soft palate.



FIG. 56
Waters' airway.

An abnormal respiratory sound, having elements both harsh and vocal, is associated with an epiglottis which is sucked on to the laryngeal aperture by each inspiration. Laryngoscopy is indicated. The epiglottis must be picked up with the beak of the laryngoscope and an endotracheal tube passed. A special instrument (Soper's laryngoscope) has been designed to facilitate

this procedure. The obstruction may be such that intubation is not needed: the amount of respiratory volume will decide.

Abnormal respiratory sounds having a vocal character suggest laryngeal obstruction. The sounds associated with laryngeal spasm are frequently high, but may be low in pitch, and occupy either one or both respiratory phases. The intensity of the sound is no guide to the amount of obstruction.

Intubation, when the laryngeal reflexes are active, may overcome obstruction, but the intercostal muscles may keep in spasm until the anæsthesia can be deepened. The more rapidly this can be effected the better. With intravenous anæsthesia this is easy; with inhalation it is difficult, because the obstruction delays the absorption of the drug.

Laryngeal spasm may be due to surgical stimulation under light anæsthesia; inhalation of too high a concentration of anæsthetic or the presence of an airway when the pharyngeal reflexes are active. When increasing the depth of anæsthesia with intravenous drugs, care should be taken not to overdose. The addition intravenously of one of the muscle-relaxing drugs would materially assist the experienced anæsthetist but they should not be used for the obstruction of organic disease.

VOMITING

No harm, great harm, or even death may result from vomiting during anæsthesia (*see page 35*).[†] Prompt treatment can avert disaster. If the laryngeal reflexes are active (which is usual during nitrous oxide-oxygen anæsthesia) the patient may protect himself by spasmodic closure of the glottis; if they are not, vomit is drawn into the trachea and he may suffocate and die, or collapse of the lung may follow.

The muscular phenomena of vomiting during anæsthesia may be obvious. On the other hand, fluid may well up into the pharynx and appear between the face and mask without any visible muscular movements or change in breathing. If a solid meal has been eaten within four hours, or liquids taken within two hours of operation, general anæsthesia is better avoided and local analgesia used, but women in labour, accident cases, battle casualties and patients with intestinal obstruction or peritonitis may have full stomachs many hours after a meal and are likely to vomit during the induction of anæsthesia.

Preliminary aspiration of stomach contents may avoid disaster. A Senoran's evacuator (*page 541*) is very useful and its tube should be left in position and aspirations repeated at intervals. If a tube is not available, vomiting should be induced by stimulating the pharynx before the induction of anaesthesia. If endotracheal anaesthesia is necessary, the use of a ballooned cuffed tube will prevent aspiration while it remains in position.

If vomiting occurs during a routine administration the patient's head should be immediately turned to one side and lowered with the jaw held forward. The vomit should be removed with gauze



FIG 57—The lateral recovery position.

until suction can be applied. This should be powerful enough to clear the pharynx rapidly. Much will depend on the promptness with which the head end of the table is lowered, but the patient must not be allowed to slide on to the floor.

Those with intestinal obstruction need gastric suction, preferably with a large bore tube, before and during the induction of anaesthesia. If a relaxant is injected with the head of the table lowered, fluids will regurgitate into the pharynx. Some anaesthetists induce anaesthesia using thiopentone and suxamethonium with the head end of the table elevated 20° , but others prefer to use an inhalation anaesthetic with carbon dioxide to stimulate deep breathing. The argument that the patient cannot breathe deeply and vomit simultaneously cannot apply in the case of regurgitation; in this case the deep inspirations are likely to favour aspiration. The essential precaution is to perform intubation as rapidly as possible and to inflate the cuff immediately.

If fluid has been aspirated into the trachea, it should be sucked out through a bronchoscope and postural coughing should be stimulated with the suction catheter. Post-operative postural coughing should be encouraged as soon as possible (*see pages 131 and 461*).

Patients left in the dorsal position are liable to aspirate vomit during the recovery period. Whenever possible they must be kept in the lateral position supported by pillows with the dependant arm behind and the head on the mattress. If the dependant thigh is straight and the uppermost one flexed (Fig. 57) the patient can be made secure with sheets.

HICCUP

The attempts of an intubated patient to hiccup interfere with surgical work particularly in the upper abdomen. They will cease if anæsthesia is deepened and this can be achieved most rapidly by further small doses of thiopentone and a relaxant. Adding ether or cyclopropane is slower and increases the explosion hazard; carbon dioxide stimulates deep breathing which also interferes with surgery.

Hiccup sometimes develops during the induction stage with thiopentone and then it is a sign that the injection has been made too rapidly. Hiccup may be an indication that the stomach is distended with fluids; gastric aspiration will relieve it.

INJURY FROM UNCONTROLLED RESTLESSNESS

During the transient restless state after a short period of anæsthesia, supervision must be constant until the patient can manage himself. After a longer period with light anæsthesia he may become restless and fall off a trolley, or in the ward he may pull out a transfusion needle or a Ryle's tube, tear off his dressings or even pitch out of bed.

Sedation can be made safe by giving a small dose first, and effective by repeating it. Morphine is probably the most widely used drug, but pethidine is analgesic and spasmolytic. Procaine by the intravenous route is effective but medical supervision has to be constant because of the danger of convulsions.

CESSATION OF RESPIRATORY MOVEMENTS BY MUSCULAR SPASM. BREATH-HOLDING

Sometimes, early during the induction with open ether, a child stops crying and holds his breath. Breathing movements cease but the chest wall is rigid. If nothing is done cyanosis soon appears. Breathing suddenly returns, often before cyanosis develops if the mask is temporarily withdrawn—a simple but effective procedure.

Lightly anæsthetised adults under cyclopropane, especially after heavy opiate pre-medication, may hold the breath long enough for cyanosis to appear. Breathing movements cease and no amount of bag pressure will conduct movements to the chest wall. Laryngoscopy reveals a closed glottis and attempts at intubation are at first fruitless. Soon a generalised pallor comes on and with it muscular relaxation. This is the moment to inflate the lungs with oxygen. Valuable time must not be lost in attempting to intubate. If the mask is firmly applied during the paroxysm, one hand can be used to exert slight pressure continuously on the bag. This will "give" slightly at the moment of muscular relaxation. Alternatively, if thiopentone can be injected, it will remove the spasm but apnoea results and artificial respiration must be continued till spontaneous movements return. Sometimes this heralds the return of breath-holding. Excess of oxygen given at such a time helps until the phase passes.

A surgical stimulus applied during light anæsthesia may cause breath-holding, but normal breathing will follow when an adequate depth has been attained. With intravenous anæsthesia this is easy, but the breath-holding itself delays the absorption of drugs given by inhalation. In such a case the exciting stimulus must be removed, when breathing will become established after a short interval. During this time no attempt should be made to increase the concentration of the drug inspired, for it will be fruitless with ether and dangerous with chloroform. As soon as breathing begins the concentration should be built up gradually.

CESSATION OF RESPIRATORY MOVEMENTS WITHOUT MUSCULAR EFFORTS—APNŒA

Cessation of respiratory movements from an overdose of any anæsthetic will soon result in asphyxia and death if artificial respiration is not immediately started and maintained. It should be easy to perform artificial respiration as the muscles are flaccid. The simplest method in the operating theatre is regular manual compression of a rubber bag filled with oxygen which is led directly into a mask (Fig. 58). This may be lifted to allow for expiration or it may be kept in position with the expiratory valve partially open provided the flow of oxygen is sufficient to keep the bag reasonably filled. The experienced anæsthetist always ensures

that plenty of oxygen is available and that a cylinder key is to hand. If the oxygen should run out then mouth-to-mouth artificial respiration must be applied (*page, 549*), or any of the methods described on *pages 543 et seq.* carried out.

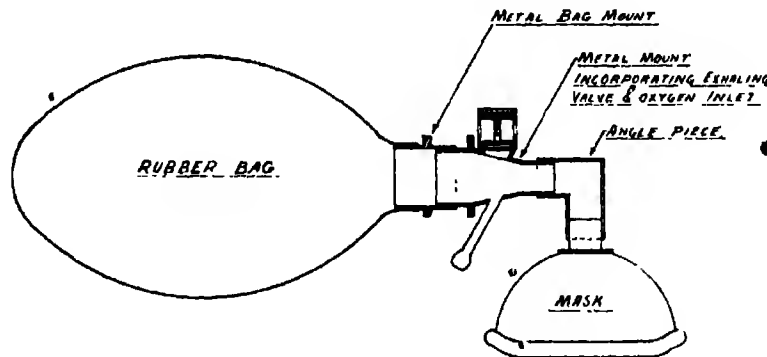


FIG. 58—Apparatus for bag ventilation technique for artificial respiration. A metal canister (Waters) containing 6 - 8 mesh soda lime, connected between the bag and expiratory valve mounts, reduces the quantity of oxygen required per minute to a minimum.

Prolonged apnoea may result from either under or over ventilation and these states are best corrected. Otherwise short periods with 10 per cent. CO_2 inhalation without the soda lime canister; deflation of the endotracheal cuff and the application of suction to the trachea may be tried with the idea of stimulating the patient to breathe.

For apnoea due to interference with muscular activity by curareiform drugs an antidote may be indicated, but until this is effective artificial respiration remains an absolute necessity. There are two kinds of interference. Acetylcholine is released when the nerve impulse reaches the motor end plate and, through its action on certain muscle receptors, produces an electrical depolarisation which causes the muscle to contract. An enzyme, cholinesterase, rapidly hydrolyses the acetylcholine. Theory has it that the large molecule of a muscle relaxant drug obstructs the path of acetylcholine to the receptors and this is termed "block by competition." The second type of block is caused by a state of electrical depolarisation similar to that produced by acetylcholine but differing in that it is prolonged.

Neostigmine reverses the effects of d-tubocurarine chloride (tubarine, syncurine), the di-methyl ether of d-tubocurarine chloride, gallamine triethiodide (flaxedil) and laudolissin, all of

which act by competitive block. Neostigmine also increases the secretions in the upper respiratory passages and atropine is frequently given intravenously along with it. Instances of sudden deaths of a circulatory type have been reported to have occurred soon after neostigmine and atropine injections. The disorder is presumably due to a summation of parasympathetic stimulation (a central action of atropine). Hunter recommends that patients should be carefully watched for at least ten minutes after these drugs have been given and if the pulse rate progressively slows he suggests that a further dose of atropine (at least 0.65 mg. (gr. $\frac{1}{100}$)) be given rapidly into a vein.

Decamethonium iodide (C. 10), suxamethonium chloride (Scoline), suxamethonium bromide (Brevdil M) and suxethonium bromide (Brevdil E) are muscular relaxant drugs which act by depolarisation block. *Neostigmine should not be used after these drugs because it destroys cholinesterase* which allows acetylcholine accumulation with further prolongation of the depolarisation. Fortunately the action of these drugs is shorter than those of the preceding group and an antidote ought not to be required; nevertheless, artificial respiration must be continued as long as necessary.

Prolonged apnoea may result from either group of relaxants, though the causes are different. The value of the test dose of a muscle relaxant drug is questioned by Churchill Davidson (1955, *Proc. Roy. Soc. Med.* 48, 622), who found that patients with localised myasthenia gravis react normally to d-tubocurarine but are resistant to decamethonium. When the disease is generalised apnoea may develop with any relaxant, therefore these drugs are best avoided at this stage.

Overdose with relaxant drugs is probably the commonest cause of prolonged apnoea. This can be avoided by knowing the effects of a small test dose and by limiting the extent of subsequent doses. Some prefer to use anaesthetic agents instead of relaxant drugs to secure muscular relaxation towards the end of operation.

Cholinesterase deficiency may occur in states of starvation, in liver disease, in poisoning by certain insecticides and possibly also in states of hypoglycaemia. There are also patients, otherwise normal, who have a reduced level of cholinesterase in the blood. The subjects of these disorders may develop prolonged apnoea after injections of depolarising agents, necessitating artificial ventilation till a satisfactory respiratory function

returns. Judgment as to when to allow the patient to breathe unaided can be facilitated by estimating the tidal volume (it should be at least 400 ml.), by diverting respirations into a bag of known volume or into a spirometer. Churchill Davidson has shown how there may be a dual type of neuromuscular block. Prolonged apnoea due to a depolarising drug may be continued by a block which can be reversed by neostigmine. There is always the danger, however, that the apnoea may be prolonged by neostigmine and artificial respiration may then have to be continued longer. Large doses of procaine should be avoided when relaxants are used because procaine, like botulinus toxin and calcium deficiency, interferes with the liberation of acetylcholine.

Deep trichlorethylene anaesthesia results in rapid shallow respirations, and if pethidine is injected at this stage, the respiratory rate is likely to decrease leading to a severe degree of respiratory acidosis. Pethidine may, however, be given to patients having trichlorethylene analgesia.

Respiratory movements may be abolished as a result of a high spinal anaesthesia, if the drug used is allowed to ascend to beyond the 3rd cervical segment of the cord. Such cases should be given artificial respiration with oxygen, using the bag ventilation technique or, failing this, by the methods described on *pages 543 et seq.* A vasoconstrictor drug (Pholedrine Sulphate B.P.C. 5 mg. intravenously) should be given if the blood pressure is low. The head-down position may cause the local anaesthetic solution to spread cephalwards and also allows gastric regurgitation. If this position is indicated to assist the cerebral circulation the patient should be intubated first with a cuffed endotracheal tube. The same applies to massive spinal anaesthesia from the inadvertent injection via the sacral hiatus of a large volume of local anaesthetic drug into an abnormally low subarachnoid space.

CIRCULATORY COLLAPSE AND LOW BLOOD PRESSURE

The cause of low blood pressure at operation may be difficult to determine since several factors may be acting simultaneously.

The anaesthetic itself and particularly chloroform may lower blood pressure. Two to three per cent. chloroform in air is safe. Stronger concentrations such as occur after breath-holding may cause collapse. On no account therefore should chloroform be dropped on a mask unless the patient is breathing.

A surgical stimulus under light chloroform anæsthesia may precipitate a similar response, the classical case being a young fit adult undergoing a trivial operation. He suddenly becomes pale, the radial pulse cannot be felt, heart sounds are inaudible, respirations become sighing and later cease. Death may occur in a matter of minutes. Similar collapse may also be caused if adrenaline is administered during chloroform, cyclopropane or trichlorethylene anæsthesia (*see page 44*). If the operation is not complicated by severe hæmorrhage, the blood pressure will usually return to normal rapidly as the administration ceases. By itself hypotension due to anæsthetic agents in normal dosage does not seem to be harmful but it may mask other effects. It requires no special treatment. The anæsthetist would be wise, however, to keep the head of the table slightly lowered and to be particularly careful to ensure adequate oxygenation and a free airway.

A large dose of a barbiturate may cause sudden circulatory failure. What constitutes an overdose is problematical for the effects vary in each patient. Single doses of from 0.5 to 1 gramme of thiopentone are frequently given to fit subjects but may well prove immediately fatal for those who are ill. Barbiturate anæsthesia can be safely conducted with small increment doses (of the order of 0.1 gramme thiopentone), after the dose required to produce loss of consciousness. Apart from the usual contraindications the administrator should not attempt this form of anæsthesia in children, patients of small stature, febrile patients, cardiac cases, or in the aged, until considerable experience has been gained.

If a sudden cardiac failure is suspected, the anæsthetist should immediately warn the surgeon and administer 100 per cent. oxygen with artificial respiration if necessary. Shoulder rests should be inserted or the patient held by the legs and the table put into a steep Trendelenburg tilt. This should not be maintained longer than necessary and the table should be gradually levelled as soon as the dilated pupils contract. If no vascular pulsation can be detected, a curved Langton Hower cardiac puncture needle (*Fig. 59*) should be inserted (after swabbing the skin with spirit) into the third right intercostal space near the sternum (*see page 20*) and passed downwards, backwards and inwards into the right auricle. This may stimulate the heart to beat again but, if it does not, the surgeon should immediately perform cardiac massage.

Various drugs have been injected into the heart such as adrenaline, strophanthin, nikethamide, digitalis, camphor, strychnine and dextrose. The successes claimed for these drugs may be due to the mechanical effect of needling the heart. Sometimes when the beat is re-established the ventricles are seen to be fibrillating. A

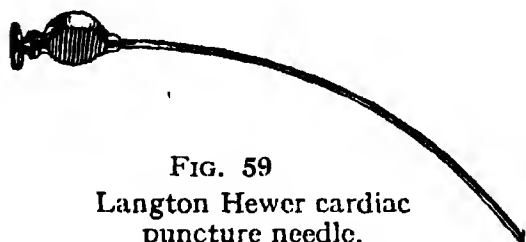


FIG. 59
Langton Hewer cardiac
puncture needle.

shock from a defibrillating apparatus may result in a more normal cardiac action. Transfusions into the brachial artery may help because the resulting rise of arterial pressure tends to close the aortic valves

and so facilitates entry of blood into the coronary arteries. Whole blood or one of its substitutes may be used. Kemp (*Brit. Journ. Anaesthesia*, 1933, 10, 158) recommends, as a last resort, the use of calcium chloride in cases dying upon the operating table. He suggests adding 0.25 ml. of saturated calcium chloride



FIG. 60—A rotary transfusion pump as used in the Royal Victoria Infirmary, Newcastle-upon-Tyne.

solution to 1,000 ml. of physiological saline at body temperature. Twenty-five millilitres per kilo. body weight should be used and 1 ml. of 1 in 1,000 adrenaline chloride solution added into the tubing of the perfusion set. It would be wise to omit the adrenaline when chloroform, trichlorethylene or cyclopropane have been used (*see page 44*). The solution must be given under pressure using a Higginson's syringe or by gravity using a funnel 1.82 metres ($6\frac{1}{2}$ feet) above the heart level. The rotary hand pump (Fig. 60) is a

recent introduction. It can deliver 500 ml. in three minutes. An additional procedure is the application of bandages from the distal to the proximal parts of the limbs.

COLLAPSE DURING CONTROLLED HYPOTENSION

Lowering of blood pressure by drugs (C.5, C.6 and trimetaphan (Arfonad, Roche)) and high spinal anaesthesia together with elevation of the operation site will reduce bleeding at operations. Should circulatory collapse ensue the best remedies are the Trendelenburg position, transfusion of blood and a vasoconstrictor drug. 1-noradrenaline (Levophed, Bayer) should be used in an initial dose of 0.1 microgramme per Kg. body weight. 4 ml. (=4 mg.) of Levophed are added to a litre of physiological saline which is given through a calibrated drip device. About 2 to 4 microgrammes per minute are necessary to maintain blood pressure, which must be measured every few minutes. Since hypotension is increased by controlled respiration this procedure should be avoided when using hypotensive drugs.

COLLAPSE DUE TO SPINAL ANÆSTHESIA

Circulatory collapse under spinal anaesthesia may be due to an abnormal mental state, to an unusually high ascent of the anaesthetic drug in the subarachnoid space, or to the response of an unstable circulatory system to moderate analgesia. Hæmorrhage may be the major or an added cause.

The patient may have concealed a natural repugnance to being "present at his own operation." The aroma and sounds of the operating theatre, coupled possibly with a fear which magnifies trivial happenings and transforms them into portents of tragedy, may combine to precipitate a type of circulatory collapse characterised by a sudden onset with facial pallor, slow pulse, low blood pressure and loss of consciousness. If the patient has confidence in the surgical team and has had adequate sedative preparation this state may never arise. If it does it should respond to oxygen, a head down tilt and the intravenous injection of a vasoconstrictor drug (Pholedrine Sulphate B.P.C. 5 mg.). A light general anaesthesia would be a humane consideration as an addition after recovery.

Spinal analgesia should not be attempted by those not trained in its use. Great care must be taken to limit analgesia to the

operation field. If the drug should inadvertently pass so high in the subarachnoid space as to paralyse breathing, artificial respiration and oxygen inhalation should be used and an injection of a vasoconstrictor drug given if the blood pressure is low. It would be wise to pass a cuffed endotracheal tube and to aspirate the stomach contents. The table should then be put into the Trendelenburg position.

Certain patients seem predisposed to collapse during spinal anaesthesia and include those with abdominal distension from pregnancy, intestinal obstruction, abdominal tumours, ascites, and obesity.

Transfusion of blood may be necessary in cases with hæmorrhage or of saline when there is dehydration. Some prefer not to use vasoconstrictor drugs and rely instead on adequate oxygenation, the Trendelenburg position and intravenous transfusion. There is no doubt that pressor drugs have only a transient effect. In the case of adrenaline this can be prolonged by giving it in dilute form continuously, adding 2 ml. of 1 in 1,000 adrenaline to 500 ml. of physiological saline (= 1 in 250,000 solution). A transient effect is sometimes needed particularly at the end of the operation to tide the patient over till he reaches the ward. These drugs should be avoided if hæmostasis has been imperfect during operation.

Some years ago it was taught that, if the systolic blood pressure fell to 80 mm. Hg. and remained at this level or below for more than 20 minutes, the patient was likely to die within 24 hours. That this is not true is shown by many patients who go for long periods with low blood pressures and survive. In the author's view, a low blood pressure of about 80 mm. Hg. is not a grave sign, although it should put the anaesthetist on his guard. He should assess the likely causes, treat the patient accordingly, and should not allow sudden postural changes. A systolic pressure of 40 to 50 mm. Hg. however, should be seriously regarded until events have proved otherwise, even though other signs indicate that all is well.

LOW BLOOD PRESSURE DUE TO SURGICAL MANIPULATIONS

Low blood pressure may immediately follow surgical manipulations such as traction on the gut or movement of an injured limb. Many procedures are without effect and what appears to

be approximately the same stimulus will lower blood pressure in one patient but not in another. The previous state of the patient may be the determining factor. One who has lost blood or who has a severe infection is specially liable to deteriorate. At operation the anæsthetist should know at what time the blood pressure became low. If it fell after the induction but before the operation began, the anæsthetic is the cause. Such low blood pressure will usually rise again without special treatment and need not cause alarm, unless very low (50 mm. Hg.). The anæsthetist should make careful records of the blood pressure during serious operations, and in time he will come to know what to expect in those who recover or deteriorate. He will soon learn that facial colour is no guide to the level of blood pressure. If low blood pressure had suddenly developed without any significant loss of blood before or during operation, then it is likely that the hypotension has been caused by the surgical manipulations, especially if the blood pressure was normal after anæsthesia became established. If the low level persists, the blood pressure will rise naturally if surgical manipulations cease. This may coincide with the end of operation; if not, a definite pause in the operation should be made. Particular care should be taken of the patient towards the end of operation as, not infrequently, several factors, such as changes in the position of the table or of the patient on the table, or the removal of inhalational apparatus, cause further lowering of blood pressure.

BLOOD LOSS AT OPERATION

The anæsthetist should note the number of swabs which are soaked with blood during operations and accustom himself to making a mental note of the amount of blood on them. He may then assess roughly how much has been lost in terms of fractions of a pint. Patients who are likely to bleed should be transfused slowly before operation and the drip speeded when the loss occurs at operation. Fit patients readily tolerate the loss of one pint of blood at operation if none has been shed previously (*see also page 48*).

Small blood losses in old and anæmic patients may result in circulatory collapse. Persistent low blood pressure at or after operation should make the anæsthetist first consider how much blood has been lost.

Obstruction to the cardiac venous return by artificial respiration with intermittent positive pressure can be reduced by using a pulmoflator, which causes a negative pressure phase during expiration. This method is indicated when artificial respiration is necessary in patients who have an unstable circulation from blood loss.

COLLAPSE AT THE END OF OPERATION OR IMMEDIATELY FOLLOWING

The patient may have collapsed as a result of the operation, and changing his position on the table or raising the end of the table may cause further deterioration.

The anæsthetic itself may have altered the clinical signs so that the patient appears to be in good condition with normal blood pressure and good colour. On withdrawing the anæsthetic, signs of blood loss or the effects of surgical manipulations may become manifest. Some of the facial pallor after operation may be due to vasoconstriction, a reaction to vasodilatation caused by the anæsthetic.

There is, therefore, a need to keep patients under close supervision during the immediate post-operative period. That the patient should spend this in the corridor under the care of a porter or junior nurse is much to be deprecated.

INADVERTENT INTRA-ARTERIAL INJECTIONS OF THIOPENTONE (*see page 18*)

CONVULSIONS DURING ANÆSTHESIA

The number of theories about the cause of "ether convulsions" bears testimony to our lack of knowledge. Nevertheless it is highly probable that convulsions can be avoided, since some experienced anæsthetists have never met them.

The stage is set for convulsions when deep ether anæsthesia is administered in a hot theatre on a warm day to a pyrexial, over-atropinised child, who has a septic focus. Warmed ether would probably accelerate the process. The substitution of some other form of anæsthesia like thiopentone or cyclopropane and oxygen, would be wise under the above conditions.

The convulsions begin in the small muscles of the face and then spread to the rest of the body. The mask should be removed, the head raised, and thiopentone injected intravenously

in doses of from 1 to 5 ml. of a 5 per cent. solution, *i.e.*, 0.05 to 0.25 G. After an interval of at least one minute, further doses of 1 to 2 ml. at a time should be given until convulsions cease. Artificial respiration may then be required. This treatment is said to be remarkably successful but there should be no delay in making the injection, otherwise the convulsions will interfere. Convulsions have also been reported during the recovery period in children after vinesthene inhalation. These are not serious and call for no special treatment.

In contrast to ether convulsions, "ether tremor" is characterised by clonic movements mainly affecting the limbs. Found usually under light anaesthesia, it is not a serious condition and disappears when anaesthesia deepens.

Fatal convulsions may occur from the use of local anaesthetics (*page 23*). They are not confined to any one particular drug, and have resulted from surface application and from infiltrations. Cocaine should be avoided in children and one should always be most careful when administering any local anaesthetic to ensure that the dose given is what is intended. Doses should be kept within the values normally used with safety. If convulsions occur, thiopentone should be injected intravenously at once and oxygen administered.

The convulsions which result from asphyxia are terminal phenomena and may be avoided by giving oxygen.

PNEUMOTHORAX AT OPERATION

When the pleural cavity is opened, the lung collapses and the mediastinum moves across to compress the other lung. During inspiration air enters both by the glottis and by the hole in the chest wall and the larger the latter the less will be the air entering by the glottis. On expiration, air from the sound lung passes partly into the trachea and into the collapsed lung. Thus the affected lung expands with expiration and collapses with inspiration (paradoxical respiration) and the patient breathes partly vitiated air. The to and fro movement of the mediastinum causes intermittent obstruction to the great veins, thus interfering with cardiac action (mediastinal flap).

The anaesthetist can lessen these changes by ensuring an adequate depth of anaesthesia with quiet breathing at the time when the pleural cavity is opened. Deep breathing must be avoided. The disorders may be dealt with either by (1) assisting inspiration

by the regular compression of the reservoir bag of the CO₂ absorption apparatus and using oxygen; or by (2) abolishing spontaneous respiration with more depressant drugs (thiopentone), and controlling respiratory movements entirely by bag compressions. When the bag is compressed (a pressure of only about 5 to 8 cm. of water is needed) both lungs expand, and both contract when the pressure is released. The exposed lung should only be expanded to approximately one third of its full capacity.

If CO₂ absorption apparatus is not to hand Boyle's machine can be used. The expiratory valve must be partially open, and sufficient nitrous oxide and oxygen should be flowing to keep enough gas in the bag to allow for intermittent compressions.

The patient should not be allowed to breathe spontaneously against a constant positive pressure; otherwise his condition will progressively deteriorate.

PNEUMOTHORAX AFTER THORACTOMY

When the chest wall has been sutured after a thoractomy some air remains in the pleural space and a corresponding amount of lung is collapsed even though positive pressure inflation has been used during the final part of closure. If nothing more is done respiration may become embarrassed and circulatory troubles follow. To counter the effects of the pneumothorax, a needle should be inserted into the pleural space and suction applied until a negative pressure of from 8 to 10 cm. of water is created.

To prevent a pneumothorax the surgeon may close the chest wall leaving an intrapleural catheter through a separate stab hole and connected with a tube (of at least 0.5 cm. diameter) dipping 2.5 cm. below the water level in a bottle of at least 15 cm. diameter. The bottle has an air outlet and should be kept 100 cm. below the level of the patient's chest. When the lungs are inflated air passes from the pleural cavity out through what amounts to a one-way valve. The patient should not be returned to the ward till X-ray examination shows that there is no mediastinal displacement. If these measures do not bring improvement the case should be reviewed. Hæmorrhage from the lung should be apparent from the presence of blood in the intrapleural tube. Bronchial obstruction may be removed by postural coughing or by broncho-

scopic suction—a practice which may be avoided if the anaesthetist employs tracheo-bronchial suction before extubation. Bronchoscopy should only be undertaken by those skilled in the procedure with the full knowledge of the dangers involved. One of these appears to be related to sudden changes in alveolar carbon dioxide tension at the period when the anaesthetic inhalation is discontinued.

HYPOTHERMIA

The ability of tissues to withstand ischaemia can be prolonged if their metabolism is depressed by reducing body temperature. Several states of emergency may result. In adults at temperatures below 27°C., and in children below 18°C., there is danger from ventricular fibrillation. This is more likely with spontaneous than with artificial respiration. Adequate ventilation by artificial respiration is therefore preferable. Additional reasons are that apnoea may develop at temperatures between 30°C. and 24°C., and the resulting fall in pH may bring on ventricular fibrillation. Electrocardiographic monitoring is essential to detect precursor conditions, *e.g.*, ventricular extrasystoles or a secondary wave following the S-wave (which indicates a fall in pH). The treatment of fibrillation is immediate cardiac massage and electric shocks with a defibrillating apparatus.

During the re-warming period a metabolic acidosis develops. This can be treated by giving a 5 per cent. sodium bicarbonate intravenously. The total quantity is calculated from measurements of the CO₂ combining power before and after cooling.

Hyperglycaemia may result if the hypothermic patient is given a 5 per cent. glucose infusion, and the plasma electrolyte concentration may fall from dilution with extra cellular fluid. It is recommended that 2.5 per cent. glucose be given and the temperature of the solution should be the same as that of the patient. Blood loss is liable to occur because of the increased coagulation time and must be made up.

Lastly, prolonged hypotension with a "failure to re-warm" has been described by Huguenard (*Anaesthet. et Analg.*, 1953, 10, 16). Injections of atropine (1.2 mg. (gr. $\frac{1}{50}$) to block the cholinergic muscarinic effects, and neostigmine (5 mg.) to stimulate the pre-ganglionic fibres are recommended with a view to liberating adrenaline. If these measures are unsuccessful 1-noradrenaline (1/250,000) should be tried.

MISUSE OF APPARATUS

Not only the colour but the labels of gas cylinders should be observed as a routine and the connections between the cylinders and flowmeters scrutinised. A reserve tank of oxygen should

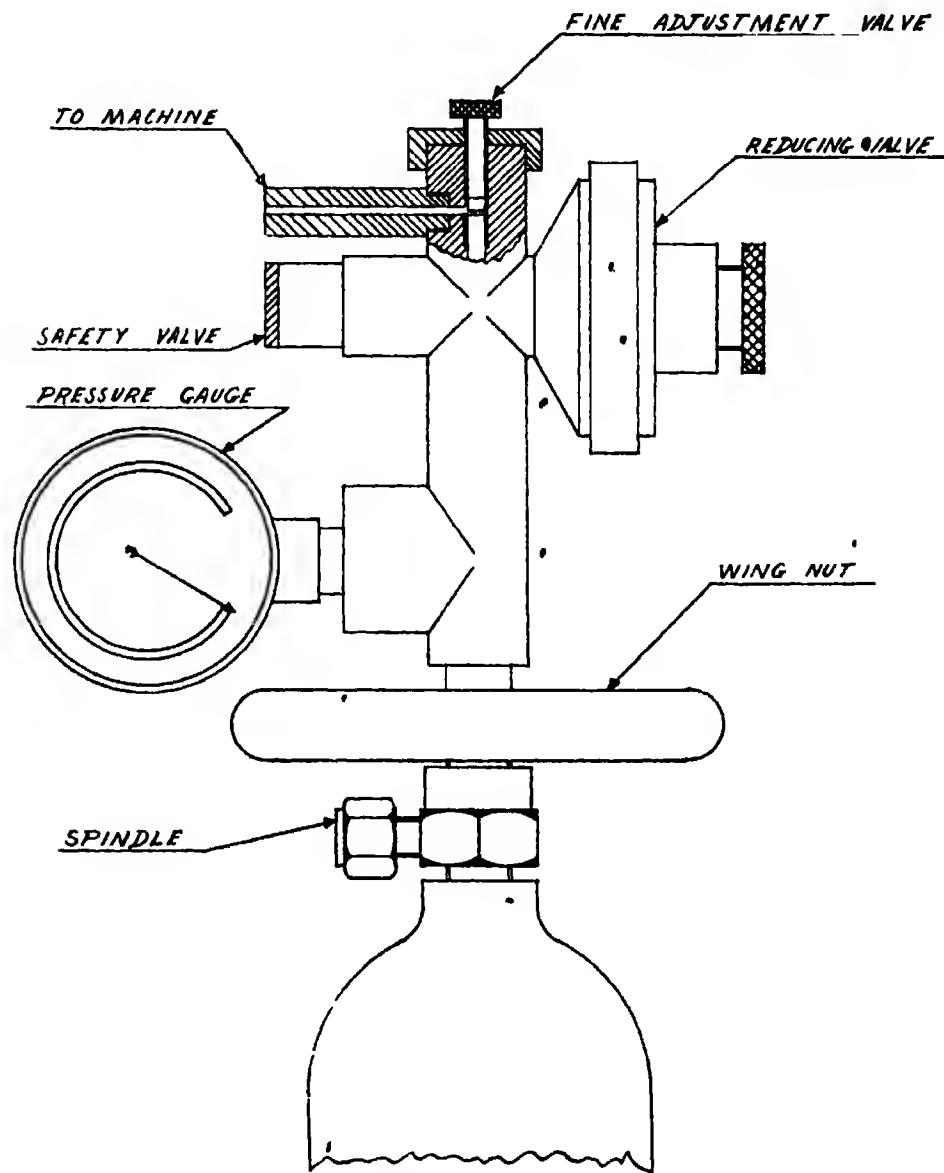


FIG. 61—Showing arrangements of valves on oxygen cylinder.

always be connected ready for use and the gauge reading checked. Emergency oxygen devices should be tested and a good flow ensured. (For danger of fire *see page 33* and for British Standard colours for medical gas cylinders *see plate facing page 33*).

The signs of oxygen deficiency which can be readily observed are cyanosis of the face with dark blood in the operation field

and rapid breathing. Later comes the terminal stage of dilated pupils, ashen grey colour, gasping irregular breathing, slow pulse and finally convulsions and death.

Inhalation of an excess of carbon dioxide causes a characteristic change in breathing, which becomes deep and rapid. The whole of the patient's effort appears to be concentrated on breathing. The skin is flushed and warm and there may be sweating, and the pulse is full and bounding. Later respiratory and cardiac failure appear. Naturally, if any of these errors are detected, the machine should be disconnected and the patient given air till they can be remedied.

Some oxygen reducing valves are surmounted by a fine adjustment screw (an arrangement which should be avoided with intermittent flow machines). It ought to be fully open since otherwise the patient will get no oxygen from a full cylinder *even though plenty of oxygen is registered by the percentage dial on the machine* (Fig. 61).

In certain types of combined apparatus either the partial rebreathing or carbon dioxide absorption principle may be used as in the Boyle apparatus with a Coxeter-Mushin carbon dioxide absorption unit. The anaesthetist who uses this equipment should take particular care to see that, when using the latter principle, the flowmeters of the Boyle unit are connected up to it, otherwise the patient will lack oxygen.

When using Boyle's apparatus the reserve oxygen cylinder should be brought into the circuit *before* the gauge on the cylinder in use registers nil. Otherwise it may empty itself when the anaesthetist's attention is elsewhere.

Carbon dioxide absorption apparatus should not be used in patients who are inhaling, or who have recently inhaled trichloroethylene (analgesia in labour), otherwise toxic and inflammable products (trichloroacetylenes) may arise which cause cranial nerve palsies.

The old Junker's inhaler contains a device so that air can be blown through the apparatus in one direction only. In the original, if the bellows was connected up to the wrong end, liquid anaesthetic could be blown into the patient. The apparatus should always be tested before use. If fluid is blown in to the patient, the anaesthetist should at once cease blowing, remove the Boyle Davis gag if this is being used, and turn the patient

over on his side. The head end of the table should then be lowered, and suction applied.

Chloroform has been given instead of ether by the open method. The anæsthetist has been surprised to find that the patient became pale and deeply anæsthetised with a small quantity of anæsthetic. The smell of the fluid should prevent this error. The treatment is to remove the chloroform and mask, and if the breathing has stopped, perform artificial respiration with oxygen.

Finally, the anæsthetist should be careful not to inject the wrong solutions. Spirit has been given instead of procaine, because both have been poured into identical containers before the anæsthetist was ready. All drugs that are to be used must be taken from labelled containers in view of the anæsthetist, and checked by him. To avoid mixing up substances used for injection many anæsthetists enclose the needle of the charged syringe by the labelled glass ampoule from which the solution has been aspirated.

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CHAPTER XXV

Post-Operative Medical Emergencies

THE clinical problem presented by the patient who, within a few hours or days of an operation, becomes acutely ill is often difficult to solve. Correct management can only be learned by experience and here all that is attempted is to collect together the possibilities and to indicate the broad lines of differentiation and treatment. Experience will bring the realisation that more than one pathological process may be threatening life. The patient is the entity and not his disease and an intangible factor, namely his will to recover, often enables him to surmount the emergency.

Many urgent post-operative happenings are within the surgeon's province and the physician is only called when something is thought to be wrong "medically." Even so, the physician should always consider whether something directly connected with the operation can be responsible, however reassuring the surgeon may be about what he has done. He should also be quite clear about what the surgeon did and particularly if the findings were negative.

The following possibilities should be considered: —

Is it pulmonary?

"Cough and sputum."

Atelectasis.

A small pulmonary embolus (*page 133*).

"Pneumonia"—Lobar.

Following atelectasis.

Inhalation abscess.

Pyæmic abscess.

Is it cardio-vascular?

Coronary thrombosis (*pages 53 and 165*).

A large pulmonary embolus (*page 133*).

Circulatory overloading (*page 48*).

Shock (peripheral circulatory failure) (*page 152*).

Thrombosis of leg veins.

Is it related to the fluid intake and output?

Dehydration—Simple water depletion.

Salt depletion and extra renal uræmia.

Potassium depletion.

Transfusion reaction (*page 45*).

Is it directly related to the operation or anæsthetic?

Toxæmia.

Fat embolism (*page 135*).

Inhaled material (*page 35*).

Pneumothorax (from misplaced paravertebral block).

Is it caused by some underlying disease?

e.g. Addison's disease. Diabetes. Thyrotoxicosis. Chronic nephritis.

PULMONARY COMPLICATIONS

These should be suspected when pyrexia following laparotomy cannot be explained by the abdominal condition. In many cases "cough and sputum" or "chestiness" is about as accurate a diagnosis as can be made.

Post operative bronchitis does occur, but it is mixed up with and part of the cause, of the commonest acute complication, namely, **atelectasis**. This comes on characteristically, 36 to 48 hours after operation. Depression of the cough reflex by drugs, upper respiratory tract infection and an abdominal wound which makes breathing painful, all play a part in its causation. The early signs are basal dulness and distant bronchial breathing resembling that heard over fluid. The auscultatory signs will vary according to whether or not the bronchus is completely blocked at the time of examination. They are not very marked as a rule because the collapsed lobe is tucked away against the vertebral column and signs arising in it are obscured by the surrounding lung. The fact that the trachea deviates from the mid-line (a difficult sign to elicit) will point to mediastinal displacement. When the heart is deviated to the affected side the diagnosis is easier.

It is claimed that intravenous procaine after operations will diminish the incidence and limit the extent of atelectasis by making respiration comfortable. It is best given by continuous intravenous drip as a 0.1 per cent. solution. For an optimal effect a dose of 4 mg. per Kg. body weight should be given in 20 minutes.

X-ray examination of the chest should always be made since the findings are of great help even if negative. In atelectasis a triangular shadow is shown at one base which represents the collapsed and usually infected, lobe. In the early stages, vascular markings may be seen crowded together in it. There will be evidence that the heart and mediastinum, diaphragm and lung fissures are all pulled towards the collapsed lobe. It is these traction effects which distinguish collapse from consolidation on the X-ray film. The intercostal spaces will be narrow. The rest of the lung is very clear from compensatory emphysema. Treatment is by massive dosage of penicillin, active movements, breathing exercises and all measures, such as expectorant mixtures and intravenous nikethamide (*see page 132*), to make the patient cough. Careful manual support of the wound by the nurse is very helpful during coughing. **Massive collapse**, a rarer and more serious condition, results from plugging of a larger bronchus (*see page 131*), but the same picture can be produced by obstruction of many smaller bronchi by excessive secretion. In massive collapse and in collapse after lung operations, bronchoscopy is sometimes, but not always, needed. If performed too late, it is not likely to help.

The term "**Pneumonia**" following operation is often loosely applied to many conditions which are more than simple "chestiness." Infection following atelectasis is the commonest. True lobar pneumonia is a rarer complication than bronchitis and atelectasis and comes on later, usually after the fourth day. Typically, there will be dulness with bronchial breathing and very often sharp, pleural pain. X-ray examination will show consolidation without the wedge-shaped shadow and traction features seen in atelectasis. Pneumonia, or a localised infection of the lung ("collapse consolidation") frequently complicates atelectasis if the plug causing bronchial obstruction is not coughed up. Pleural pain and hæmoptysis indicate **pulmonary infarction** from embolism (*see page 133*) and call for anti-coagulant therapy (*see page 595*). X-ray examination shows a wedge-shaped area on the surface of the lung. This infarcted area may break down and become an abscess. **Pyæmic emboli** may complicate operations through grossly septic tissues, *e.g.*, perinephric abscess. They show as multiple X-ray shadows in both lung fields, which quickly develop fluid levels characteristic of abscesses. Chemo-therapy and antibiotics are urgently indicated.

An inhalation, or obstructive, **lung abscess**, is an urgent post-operative complication, which sometimes arises in patients with infected gums and after tonsillectomy. An inhaled infected fragment usually reaches the most dependent part of the lung in the lying position, *i.e.*, the scapular branch of the upper lobe bronchus, or the apical bronchus of the lower lobe, and causes an area of atelectasis with subsequent necrosis. Because the right main bronchus is more nearly vertical than the left, inhaled particles are more likely to reach the right lung than the left. X-ray examination, and particularly a lateral view, shows an area of consolidation and later a fluid level. Foul smelling sputum is usual. Postural drainage should be started at once and a thoracic surgeon consulted.

CARDIO-VASCULAR COMPLICATIONS

Both coronary thrombosis and **massive pulmonary embolism** cause pain in the centre of the chest. The picture of massive pulmonary embolism (*page 133*) is that of dyspnoea of abrupt onset in a patient in bed after an operation. The pain is not intense and is better described as suffocative distress. In **coronary thrombosis** (*pages 53 and 165*) pain is severe and collapse marked and progressive but the symptoms are less abrupt in onset than in pulmonary embolism. The differential points are further discussed on *pages 133 and 165*.

Circulatory overloading should be considered if much fluid has been transfused (*page 48*). The amount given and its rate of administration should be scrutinised (*page 154*). In a patient whose myocardium is poor, excessive transfusion after blood loss may cause the pulse rate to rise and the blood pressure to fall. In such a case these are the signs of cardiac failure and not of further hæmorrhage. On auscultation the characteristic signs are basal râles, but sometimes a patient is too ill to breathe deeply enough to produce stethoscopic signs. Other causes of "moist lungs" are acute bronchiolitis and left ventricular failure unrelated to intravenous therapy.

In deciding how much of the picture is due to **shock**, the nature of the operation, its duration, and the amount of blood lost, should be reviewed (*page 152*). Profound fall of blood pressure after extensive operations, such as **synchronous** combined excision of the rectum, should be treated as described on *pages 446 et seq.*

Thrombosis of leg veins.

This is a dangerous, if initially silent, complication which should be looked for after any operation which keeps the patient in bed for more than a day or two. There is usually mild pyrexia. Early signs are slight œdema of the foot which masks the extensor tendons and slight fulness of the foot veins. Dorsiflexion of the ankle causes pain in the calf (Homan's sign), which is also tender on pressure. Anti-coagulant therapy (*page 595*) should be started at once. Surgical advice should be sought on the question of ligation or partial occlusion of the femoral veins by a clip. These measures will prevent massive pulmonary embolism and should certainly be considered if there has been hæmoptysis and pleural pain from embolism of peripheral origin.

POST-OPERATIVE FLUID AND ELECTROLYTE DISTURBANCE

This presents either as an urgent preventive problem in a patient who is in normal fluid and electrolyte balance before operation or as a problem in treatment of an already dehydrated patient.

Prevention.—A normal 70 Kg. man needs 2 to 3 litres of water (given as tap water or intravenous 5 per cent. dextrose) and 4·5 G. of salt (one bottle of physiological saline) a day. For several days after operation less is needed because the endocrine response causes salt and water retention. As the salt output is low estimations of urinary chlorides during this period might wrongly suggest the need for extra salt but the plasma sodium level will show that the patient is full of it.

Treatment.—Post-operative dehydration is usually the result of both water and salt depletion and is shown clinically by lassitude, headache, nausea and cramp. The blood urea is high (extra-renal uræmia) and hæmoglobin concentration raised. Confusion may suggest mental illness and cramp of the abdominal wall may make one wonder whether an acute abdominal complication is present. Thirst is not necessarily marked unless there is depletion of water as distinct from depletion of salt.

The following tests are of great value in the dehydrated patient.

(1) Fantus' test for chlorides in urine.

Reagents and apparatus—

20 per cent. potassium chromate solution.

2·9 per cent. silver nitrate solution.

Distilled water.

Test tube.

Small pipette ("fountain pen filler").

N.B.—The same pipette must be used throughout the tests so that the drops shall be of the same size.

Technique—Filter the urine, if not already clear, and if albumin is present remove by boiling and filtering.

Take 10 drops of urine in the test tube.

Rinse pipette.

Add 1 drop of potassium chromate solution (the indicator).

Rinse pipette.

Add silver nitrate solution drop by drop and shake the test tube after each drop.

End point is a sharp change of colour from yellow to brown.

The number of drops needed to produce the colour change indicates the number of grammes of sodium chloride per litre.

N.B.—It is well to do a preliminary test with distilled water to make sure there is no contamination of the chromate solution with chloride.

Chlorides should be regarded as absent if the end point is reached with the first drop. This may occur in normal persons if urine is very dilute. If it occurs in concentrated urine (specific gravity 1020 or more) less than 3 G. per litre suggests salt depletion.

A patient is not suffering from salt depletion if the urine contains 5 G. or more of salt per litre (except in Addison's disease and during the administration of intravenous saline).

Special silver chromate test papers are provided on board ships for testing urine for salt. If the salt output is normal the paper quickly becomes bright yellow in contact with urine.

(2) Specific gravity of plasma.

This may be quickly and easily measured by the falling drop method. It is useful in deciding how much fluid (but not salt) is needed to correct severe dehydration. The laboratory has a row of bottles of copper sulphate solution of different specific gravities. The patient's plasma is obtained by centrifuging heparinized blood and a drop of it is released into the bottle from a pipette whose point is 1 cm. above the surface of the solution. The rise or fall of the drop during the ten seconds after it has lost its initial momentum indicates whether it is lighter or heavier than the test solution. It is well to do a control test on normal plasma at the same time. A bottle of 100 ml. of the solution may be used 100 times before its specific gravity is affected. The normal specific gravity is 1027 and for every 0.001 rise above this 200 ml. of water is lacking in the vascular compartment of body fluid (providing the plasma proteins were originally normal).

(3) Plasma electrolyte estimations.

Normal levels.

Sodium.—143 mEq/litre (range 133 to 152) or 330 mg. per 100 ml. (range 306 to 350).

Potassium.—5 mEq/litre (range 3.6 to 5.6) or 20 mg. per 100 ml. (range 14 to 22).

Chloride.—100 mEq/litre (range 99 to 107) or 355 mg. per 100 ml. (range 350 to 380).

(Sodium chloride.—100 mEq/litre (range 97 to 108) or 585 mg. per 100 ml. (range 570 to 630). In some laboratories only the chloride is estimated and the result expressed as sodium chloride).

Bicarbonate.—27 mEq/litre (range 25 to 33) or 60 volumes CO₂ per 100 ml. (range 56 to 74).

Proteins.—15 to 20 mEq/litre or 6 to 8 G. per 100 ml.

When a patient vomits or loses drainage fluid after operation his plasma sodium falls and chlorides disappear from his urine. Salt must be replaced intravenously. When much has to be given it is well to replace one bottle in three or four by M/6 sodium lactate solution to prevent acidosis. Its electrolyte content (154 mEq of Na per litre) can be regarded as the same as that of physiological saline (160 mEq of Na per litre).

A rough idea of the amount of sodium needed can be obtained by calculation as follows :—

Deficit of Na = (normal level – patient's level) × volume of body fluid

= 142 – Patient's Na level in mEq × 60 per cent. of body weight.
For a 70 Kg. man with a plasma sodium of 118 mEq/litre deficit

$$= (142 - 118 \times \frac{60}{100} \times 70) ,$$

$$= 24 \times 42 = 1,008 \text{ mEq Na (approx.)}.$$

1 litre of physiological saline = 160 mEq Na (approx.)

∴ the required replacement

$$= \frac{\text{Deficit}}{160} \text{ litres of physiological saline}$$

— about 6½ litres.

A similar formula is—

litre of physiological saline needed

$$= \frac{142 - \text{patient's serum sodium level in mEq}}{3}$$

Such a calculation is only a rough guide for it is fallacious to suppose that, knowing the body weight and the plasma electrolyte levels it is possible to calculate exactly how much salt is needed to bring the plasma level back to normal. Calculations cannot allow for the state of the intra-cellular fluid or the efficiency of the kidneys and they ignore the adjustments which are continually taking place.

We should judge the patient's state by considering all the evidence: the history of fluid loss by vomiting, diarrhoea or through a gastric suction tube or fistula; the presence or absence of thirst; the clinical signs of dehydration (slack skin, dry tongue, soft eyeballs and collapsed veins); the urinary volume, specific gravity and chloride content and the serum sodium and potassium levels.

It must be remembered too that in addition to replacement of deficits the patient must receive also his basic needs of 1½ litres

of fluid (best given as 5 per cent. dextrose) and $\frac{1}{2}$ litre of physiological saline (= 4.5 G. NaCl).

In general it can be stated that a patient with thirst and oliguria is short of about 4 litres of fluid and if his B.P. is below 90 and chlorides are absent from his urine he needs 0.75 G. NaCl per Kg. body weight (*i.e.* about $4\frac{1}{2}$ litres of physiological saline for a 60 Kg. man).

Potassium depletion.

This should be suspected after operation if there is muscular weakness, abdominal distension and mental confusion. It can be confirmed by estimating the serum potassium by flame photometry. E.C.G. changes are less reliable particularly in acute depletion (*see page 240*). In salt depletion the extra-cellular fluid is hypotonic and the body cannot easily correct this because cell membranes are impermeable. Any sodium which reaches the plasma is retained but potassium cannot be held back by the kidneys and is lost. These effects are increased if, after operation, potassium free fluid is infused for several days.

Treatment.—Potassium depletion is best corrected by giving potassium by mouth. The potassium citrate mixture of the National Formulary is suitable (Pot. Citrate 3 G. (gr. 45), Citric Acid 0.6 G. (gr. 9), Syrup of Ginger 4 ml. (m 60), Chloroform water to 15 ml. ($\frac{1}{2}$ fl. oz.)), and 15 ml. ($\frac{1}{2}$ fl. oz.) of it contains 25 mEq. of potassium. Alternatively capsules containing 1 G. of potassium chloride (13 mEq. potassium) can be used.

When a patient can take food and fluid by mouth we need not worry about his potassium intake but if intravenous fluid is needed for several days it is generally necessary to give potassium. In all cases a deficit should be demonstrated first. It is best not to give potassium until water and sodium deficits are corrected and the urinary output is good.

When intravenous replacement is needed a useful solution is NaCl 2.25 G. (= 40 mEq.), KCl 3.0 G. (= 40 mEq.), 5 per cent. dextrose 1,000 ml. Two litres of this in 24 hours will give the patient his 4.5 G. NaCl (80 mEq.) and 6 G. KCl (80 mEq.). An extra fluid needed should be given as 5 per cent. dextrose. Darrow's solution (2.7 G. KCl and 4 G. NaCl per litre) is similar.

Another plan is to inject potassium chloride into the bottle. Not more than 1.5 G. of potassium chloride should be put into one bottle since this strength (40 mEq. per litre) is the maximum safe

strength for intravenous administration. The rate should not exceed 12 mEq. per hour.

Potassium intoxication.

This is an occasional post-operative complication and results from excessive intravenous medication particularly if there is renal failure, or if potassium citrate has been given (uselessly) as a diuretic. The main symptoms are weakness, flaccid paralysis and paræsthesiæ. Sudden death may occur from cardiac arrest even when the patient does not appear very ill. The lethal level is 10 mEq/litre (40 mg. per 100 ml.). Characteristic E.C.G. changes occur.

Treatment.—Give Mer'salyl 2 ml. intramuscularly to promote diuresis. Give insulin and dextrose (0.25 unit of insulin for each gramme of dextrose). This reduces extra-cellular potassium by causing increased cellular uptake.

COMPLICATIONS DUE TO ANÆSTHESIA, OPERATION AND UNDERLYING DISEASE

In assessing the part played by the operation, the duration, amount of blood lost (*see page 152*), and degree of toxæmia, should be considered. Fat embolism (*see page 135*) after bony injury may be overlooked unless special signs are sought.

The physician should always know the details of anæsthesia employed and also exactly what the surgeon did. This may be particularly important if the findings were negative, for it is not unknown for an acute abdomen of medical nature to be opened and the true diagnosis (*e.g.*, coronary thrombosis and Addison's disease) to be made later.

Acute dilatation of the stomach.

This may be an early complication of an abdominal or other operation and also of injuries and fractures which are accompanied by shock. It may occur in the cabinet respirator (*see page 33*). The characteristic symptom is effortless copious vomiting of fluid like dirty water at a time when any post-anæsthetic vomiting should have ceased. Should it complicate gastrostomy, the typical fluid will come out of the opening in the abdominal wall. Earlier evidence is hiccup, epigastric discomfort, nausea and a rising pulse rate. When suspected, a stomach aspiration tube should be passed and the stomach kept empty by continuous suction. The patient

may drink, but the fluid must be aspirated. Physiological saline should be given intravenously. Most cases can be nursed propped up, but desperate ones should certainly be kept flat or even in the Trendelenburg position.

Peptic aspiration pneumonia (Mendelsohn's syndrome).

During an obstetric operation under inhalation anæsthesia to effect delivery there is slight (even overlooked) vomiting. All this is well for 2 to 5 hours and then the patient becomes very ill with cyanosis, dyspnœa and tachycardia. The clinical diagnosis based on extensive lung crepitations and low blood pressure is acute pulmonary œdema. The condition is thought to be specially liable to complicate pregnancy because of the sudden loss on delivery of corticotrophin and glucocorticoids produced by the placenta. The patient is unable, when the stress caused by inhaled vomit occurs, to meet the demand for these hormones and acute adrenal insufficiency results. It is therefore suggested that in addition to oxygen, antibiotics, bronchoscopy and general measures, hydrocortisone 100 mg. should be given intravenously.

Paralytic Ileus.

Vomiting, distension and absolute constipation after operation point to obstruction and although mainly a surgical problem the doctor called to this post-operative emergency should be aware of the possibilities and so these notes are added for the non-surgeon who may be called when mechanical obstruction can be excluded.

Although distension may result from involvement of the cœliac plexus by carcinoma (Ogilvie's syndrome) most cases are so-called "paralytic ileus" which often has a background of peritonitis. Sometimes the infection is not in the peritoneum but elsewhere (*e.g.*, pneumonia, *page* 142, and typhoid, *page* 314). There are also non-infective causes acting by sympathetic stimulation such as fractures, chest injuries and even catheterisation and the application of plaster jackets.

Paralytic ileus is a very serious condition because it leads to dehydration and alteration in blood electrolytes. Its early stages are signalled by a rising pulse rate and hiccup. Then vomiting, constipation and distension appear and there is silence on auscultation.

Treatment should be by intestinal tract suction by Miller-Abbott or other tube; passage of a rectal flatus tube; oxygen in high concentration (especially valuable in children); correction of

dehydration (*see page 463*) and electrolyte loss, including potassium (*see page 466*); vigorous treatment with an appropriate antibiotic and adequate sleep. Sodium phenobarbitone with pethidine by injection is a useful combination. Stimulation of the bowel by physostigmine (eserine), pituitary extracts, carbachol and Prostigmin (neostigmine) which is sometimes adopted for the meteorism of pneumonia (*see page 144*) is generally frowned upon in paralytic ileus.

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CHAPTER XXVI

Bites and Stings and Miscellaneous Emergencies

BITES AND STINGS

Mosquito bites.

THESE are best treated by a dab of spirit or other antiseptic. If a patient has reacted severely to an insect bite, he should be given ephedrine 32 mg. (gr. $\frac{1}{2}$) twice a day.

Bee and wasp stings.

While bee stings are usually trivial to the experienced apiarist, they may be serious to others. Occasionally coma and death have followed within 20 minutes. Since in these cases no local reaction has occurred and the site has been on thin vascular skin, it is assumed that the poison entered a venule by chance. It is said that bee stings on the back of the neck are liable to cause fainting from reflex vagal inhibition. Status asthmaticus has followed a sting.

Treatment.—A wasp does not leave her sting behind but a bee does, together with the poison sac, the muscles of which go on pumping in poison for several seconds. Hence it is best not to pick out the sting lest the fingers squeeze in more venom but rather to scrape it out with a finger nail or wipe it out with a handkerchief. An antihistamine cream is the best application to use. Failing this an alkali such as "blue bag," bicarbonate of soda or soap may be applied. A weak acid, e.g. vinegar, is said to be better for wasp stings.

Adrenaline is necessary for the more serious symptoms such as generalized urticaria. Antistin (antazoline), 100 mg. subcutaneously, or a similar preparation (*see page 611*) is also effective and should be used when a bee or wasp is swallowed.

Bee-keepers who know they react badly to stings may prevent serious symptoms by taking ephedrine 16 mg. (gr. $\frac{1}{4}$) by mouth before handling bees. Desensitization by injections of filtered extracts of whole bees has been suggested. The insulin requirements of a diabetic will be increased by a severe reaction to a bee sting.

Blistering beetles produce skin lesions requiring local treatment by alkaline lotions. Fluid from crushed beetles, if it reaches the

eyes, causes an acute conjunctivitis and so do the hairs of various caterpillars. Treatment is on general lines.

Fish stings.

The effects of these and their immediate treatment are not as widely known as they should be. Many fish, such as weevers and some dogfish, have sharp spines with a poison gland at the base. In the case of the sting-ray the spine is frequently broken off and left in the wound. It can be shown by X-rays and must be removed. The lesions inflicted by stinging fish are usually on a finger or on the sole. They closely resemble snake bite, and occasionally tetanus bacilli are inoculated simultaneously. The initial pain, invariably described in the strongest possible terms, is accompanied by disabling prostration, which may explain some otherwise unaccountable bathing fatalities.

Treatment.—This is on the same lines as for snake bite. The poison, a toxalbumin, is readily destroyed by potassium permanganate. A 5 per cent. solution (which is saturated and has a deep purple colour) should be injected into and along the track of the wound despite the swelling and tension of the part. Failing this, the wound should be incised and crystals of potassium permanganate rubbed in. Relief of pain is instantaneous. Alternatively, infiltration with 2 per cent. procaine may be used.

H. Muir Evans, in his book, "Sting Fish and Sea Farer," gives this vivid account of a sting by a lesser weever, which happened to his eight-year-old son.

"While bathing a few yards from his home he was stung in the foot and brought home crying and in great pain. I happened to return to the house shortly afterwards, and, having found the wound, immediately carried out the treatment as above described, using a hypodermic needle and permanganate solution. The condition of the boy was alarming as he was very pale, with a rapid feeble pulse and cold sweats. He seemed beside himself with pain and the insertion of the needle into the wound was not even noticed. Within a few minutes of the injection the pain was completely relieved, and after a glass of hot milk he was anxious to return to the beach. After an hour's rest he was allowed to rejoin his fellows; no swelling or inflammation followed the sting as its virulence was at once destroyed by chemical means."

Jellyfish, even the tiny ones, can cause a severe sting on quite fleeting contact with the numerous trigger hairs on the tentacles. A painful swollen area of erythema with a white centre quickly develops. In tropical waters the effects may be more serious and fatalities have been recorded. Lumbar pain may be a marked feature. For severe reactions cortisone 25 mg. by mouth

4-hourly, Injection of Adrenaline B.P. subcutaneously and a slow intravenous injection of 10 ml. of 10 per cent. calcium gluconate are recommended.

Spider Bites.

The bite of the "Black Widow" spider of America and related species in the Mediterranean zone, Australia, South Africa, and Russia is capable of causing severe symptoms. As the eggs are often laid around privies, a common site for the bite is the perineum or genitalia. The bite may not be felt but two small red spots are to be seen. A non-hæmolytic neurotoxin causes cramping pain all over the body, often within 15 minutes of the bite; the muscles become rigid, and an "acute abdomen" may be simulated, but there is no tenderness or rise of pulse rate. Other features are pyrexia, leucocytosis and a macular rash. A burning sensation in the soles of the feet is said to be pathognomonic. Spider bites cause a high death rate in infants but are non-fatal in adults.

No British spiders inflict severe bites but a very aggressive one (*Phoneutria fera*) which strikes on the slightest provocation is often imported on bananas. Only local symptoms result.

Treatment.—Morphine may be required. Specific anti-venine should be used if available (Mulford Laboratories, Philadelphia). The dose is 2.5 ml. and may be repeated. It is supplied dry and should be mixed with warm water. Avoid shaking as this causes troublesome foam. Failing this, a slow intravenous injection of calcium gluconate 10 to 20 ml. of 10 per cent. solution may be used and 2 ml. of 50 per cent. magnesium sulphate given intramuscularly. A.C.T.H. has been recommended.

Scorpion stings.

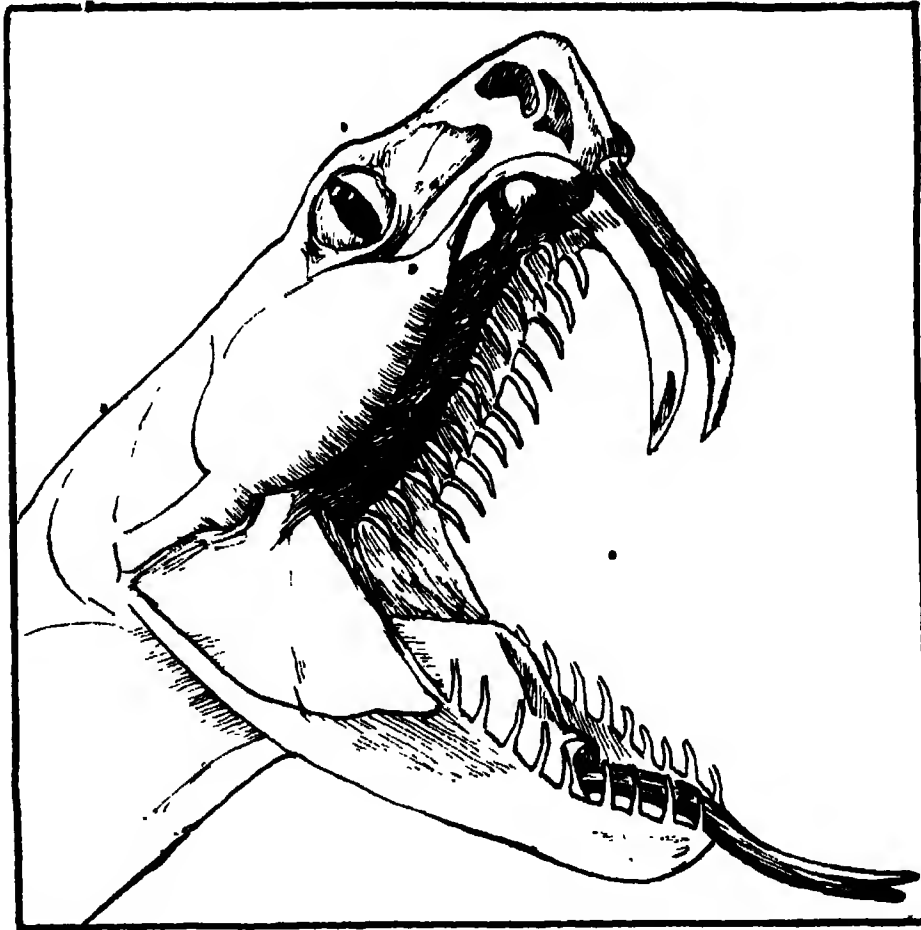
The scorpion is an arachnid about 50 mm. long, with an armoured tail ending in a large sting, the poison of which causes burning pain followed later by salivation, nausea, vomiting, drowsiness and collapse. Muscular cramps may be a feature. In young children, cardio-respiratory paralysis may result in death.

Treatment.—A tourniquet should be applied if possible and suction started. Ammonia or rectified spirit in which scorpions have been killed should be applied. Procaine, 2 per cent., by injection will relieve the pain. Specific anti-venine should also be used (Allen & Hanburys Ltd., 7 Vere Street, London, W.1.; Telephone GROsvenor 7571).

Snake bite.

Prevention.—In snake infested country never reach up and put your hand on a rock or step on to a fallen log, or approach a rough stone wall without testing out first with a long stick.

Diagnosis.—Typically two fang marks are visible but not always and the local effects may be trivial. Accurate diagnosis depends on seeing the snake and knowing the kinds which live in the



(American Museum of Natural History.)

FIG. 62

Head of poisonous snake showing poison apparatus.
Note the long tubular fangs.

neighbourhood. The only poisonous English snake is the adder or viper (*Vipera berus*). It may be recognized from its appearance (Fig. 63) but the markings may be indistinct and the colour may vary from reddish brown to black. Its length rarely exceeds 20 inches and a large snake, over 30 inches long, in England is almost certainly incapable of harm. Snake venoms are mostly powerful enzymes which have numerous effects—neurotoxic, curare-like,

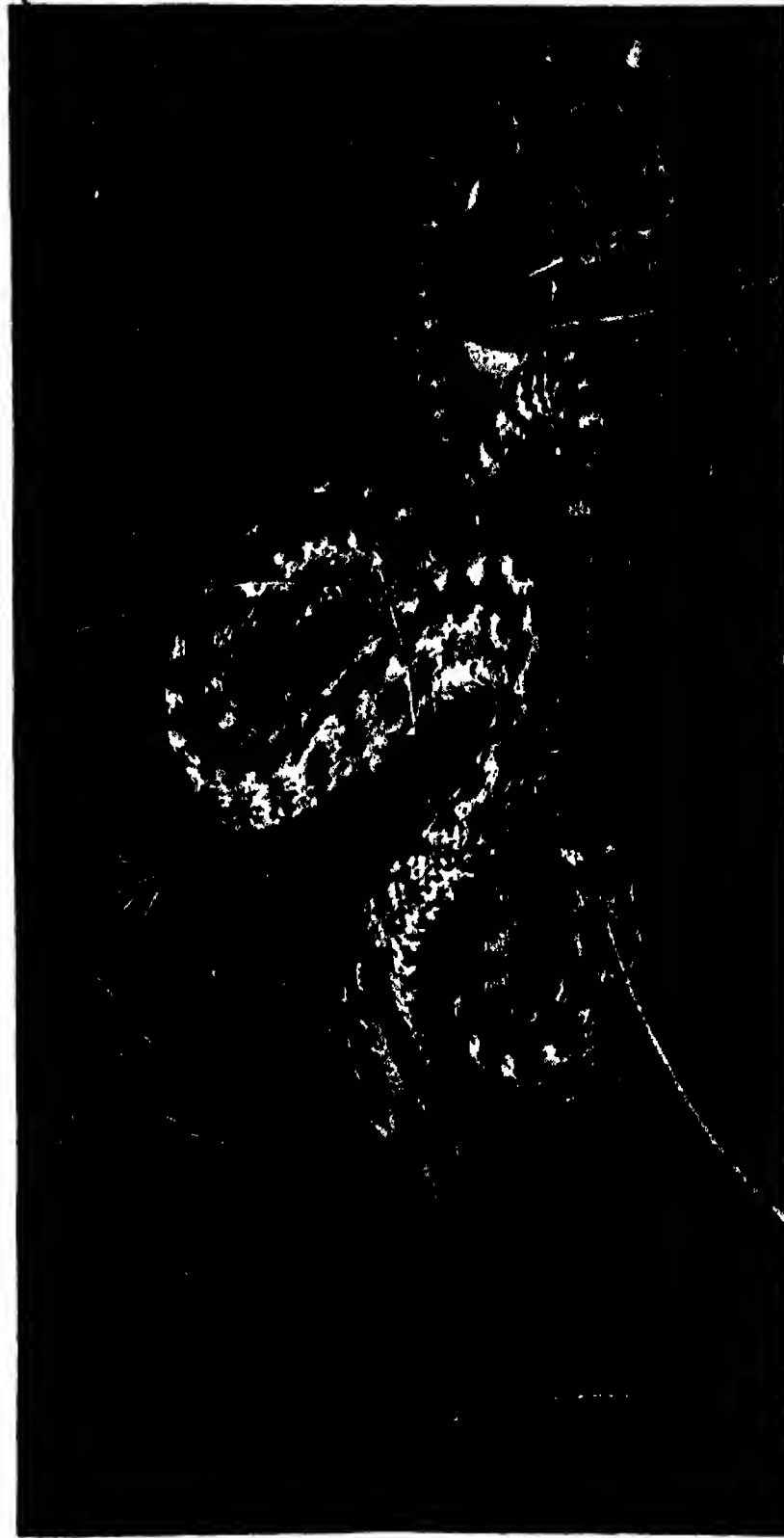


FIG. 63

The Adder or Viper (*Vipera berus*)

poison label"—the black zig-zag marking down the back

N

histamine-liberating and hæmolytic. Some enhance and others inhibit blood coagulation. The victim may show muscular weakness, vomiting, ataxia, diplopia and, later, coma or the effects may be simply local pain, swelling and discolouration. The victim is usually very frightened.

Treatment.—

PREVENT ABSORPTION OF POISON.—Place a tourniquet round the finger, arm or leg near to the bite and over the clothing if necessary. As venom is usually injected superficially and travels by lymphatics the tourniquet need not be very tight. Keep it on until other measures have been carried out. If this is longer than an hour release it for a few seconds every half hour. If ice is available it can be applied to delay absorption.

REMOVAL OF POISON.—Wash the bitten area with water, urine or saliva. If the snake is seen and is certainly or doubtfully venomous or if it escapes, excise the bitten area. Remove any broken fangs and apply suction for half an hour or so with a breast pump, preheated bottle or the mouth (but not if the lips are cracked). As many snakes dribble venom when they bite, the wound should be washed before excising. A more elaborate method of removing toxin is to put on two tourniquets—an arterial one and, distal to this, a venous one—*i.e.* obstructing the veins only. Then incise a vein draining the area of the bite and undo the arterial tourniquet for 30 seconds so that blood enters the part and, escaping from the incised vein, washes out venom. Tighten the arterial tourniquet again and gently “milk” the limb distally and proximally towards the bite, thus squeezing out blood and venom. Release the arterial tourniquet again for 30 seconds and repeat the whole process several times until, in an adult, some 600 ml. of blood have been removed.

ANTI-VENINE.—Liberal amounts of anti-venine should be injected intravenously as soon as possible into the unaffected arm or leg (*page 579*). Small people and children need a relatively large dose. (For sources of supply in different countries *see page 621*). Serum should also be infiltrated into the bitten area. As anti-venines are prepared from horse serum anaphylactic symptoms may be encountered (*page 40*). Anti-venines are highly specific and so the snake should be identified if possible. Otherwise local knowledge and the history will help to decide. If there is doubt a polyvalent serum should be used. When serum cannot be

obtained at once 1 ml. of a 5 per cent. emulsion of soap may be injected at four or five points surrounding the bite *but this is only a first-aid measure.*

GENERAL MEASURES.—Anti-histamine drugs (*page 611*) should be given. A.C.T.H. 25 units intramuscularly or cortisone 25 mg. by mouth 6-hourly is recommended. The affected part should be kept at rest and the patient should not be allowed to walk about. Warmth increases the absorption of venom and should be avoided. Cooling would be more reasonable. Because of the risk of respiratory failure morphine is contra-indicated. Alcohol should not be given unless nothing else is available. Artificial respiration (*page 543*) and treatment for circulatory failure (*page 152*) may be needed. Tetanus anti-toxin should be given.

Spitting snakes.

Certain colubrine snakes in Africa are able to eject their venom considerable distances and to aim it accurately. The patient usually sees the snake and almost immediately feels an intense burning in the eyes. Severe conjunctivitis results. Treatment consists of repeated irrigation with anti-venine, if available, or with saline, or 1 in 5,000 potassium permanganate (*i.e.*, 2 grains or 3 or 4 crystals per pint), and instillation of 1 per cent. atropine drops.

Dog Bites.

The modern view is that a dog bite should be treated like any other wound. It may be cleaned with soap and water but not scrubbed. Then it should be doused with hydrogen peroxide or strong potassium permanganate solution and covered with a moist dressing. It would be wise to give 1,500 international units of tetanus anti-toxin or, if the patient has had tetanus toxoid already, a booster dose of it should be given. Cauterisation with fuming nitric acid or pure phenol in an attempt to kill the virus of rabies is not now recommended because it is painful, it may seal organisms deep in the wound and it leaves scars. In any case, rabies is very unlikely in Britain, where no case has been contracted since 1922. In the U.S.A. the annual number of deaths from rabies from 1932 to 1952 inclusive has varied from 18 to 80 (average 44). As rabies is present on the Continent and elsewhere, cases may be expected if quarantine regulations are infringed. Persons bitten when abroad may develop the disease here, the last such case being a fatal one in a soldier in 1947.

RABIES

The disease is not an urgent problem once symptoms have appeared since it is then invariably fatal. Sodium phenobarbitone 120 mg. (gr. 2) intravenously can be used to control the distressing symptoms (Figs. 64 and 65).



FIG. 64

The bloody drooling and facial expression accompanying the pharyngeal spasm of rabies. The hand on the throat is characteristic.

The bite of a possibly rabid animal, however, always raises the urgent question of prophylactic treatment. Dogs and many other animals, including cats, wolves, jackals and vampire bats may act as vectors of the virus. Infection may result not only from biting but also from their licking fresh wounds or abraded skin. Attendants may be similarly affected by patients. Bites by fleas from rabid animals do not carry the infection. Milk and meat from a rabid animal may be consumed with impunity.

Since specific treatment is not entirely innocuous (very rarely there are neurotoxic sequelæ) the important question to answer is: "Is the dog rabid?"

A rabid dog shows changes in disposition, becoming either morose or irritable. The bark changes. The dog is easily startled and appears ill and later shows convulsions or paralysis. A rabid dog is infective for not more than four days before it develops symptoms, after which it dies within six days. When possible,



FIG. 65

The cry and accompanying pharyngeal spasm of rabies.

therefore, a suspected animal should be caged and observed, and if it survives ten days it could not have been rabid at the time of biting. Specific treatment for the victim of its bite would not be required, or if commenced could be safely discontinued.

If a possibly rabid animal has to be killed chloroform should be used and not violence. The body should be sent to the nearest suitable laboratory. If a post-mortem has been made and rabies is suspected the head and neck should be wrapped in a cloth wrung out in 1 in 1,000 corrosive sublimate and placed in a tin or box marked "Pathological specimen. Fragile with care" and sent by post (*not* parcel post) to the laboratory. Further advice

can be obtained from the Ministry of Health, 23 Savile Row, London, W.1 (Tel. REGent 8411), or the Department of Health for Scotland, St. Andrew's House, Edinburgh (Tel. WAVerley 7241).

If the animal cannot be caught and observed, or if it has been killed and there is strong reason to suspect rabies, or if the wound is on the head where nerve paths to the brain are short, it may be advisable to start treatment at once. In the case of peripheral wounds, since the incubation period varies from two weeks to several months, depending on the length of peripheral nerve to be traversed by the virus, there may be time to confirm the possibility of infection before starting treatment. Vaccine has no effect on those cases, *e.g.*, face bites, where the incubation period is likely to be less than 30 days. For these, serum (when commercially available) injected around the wound may prolong the incubation period.

Treatment consists in giving a series of injections of vaccine prepared from attenuated living fixed virus or killed carbolised or etherised virus. The amount and spacing of doses depends upon the type of vaccine used and the probable severity of the infection. In mild cases, 2 ml. of a 1 per cent. suspension of infected sheep brain in 0.5 per cent. carbol-saline may be injected subcutaneously daily for seven days. In cases of average severity 5 ml. may be given daily for 14 days. In the case of severe lacerated wounds on the neck and face, 10 ml. should be given daily for 14 days. As antibody after vaccine does not appear for 14 days, hyper-immune serum should be used as well to bridge the gap.

Human bites.

These generally occur on the hands because the closed fist is hit against the teeth. If the tendon sheath is lacerated the tendon carries infection upwards when the hand is opened. These bites should therefore be treated with great respect. Tetanus antitoxin 1,500 units should be given or a booster dose of tetanus toxoid if the patient has already been immunised.

Systemic penicillin therapy should be started at once. Suturing should be avoided and a surgeon's advice sought.

FOREIGN BODIES

Foreign body in the ear.

Unless it is a pea or bean which would swell, this is best removed by syringing (using the child's water pistol if a syringe

is not available). Otherwise, a small hook should be insinuated past the foreign body and then turned and withdrawn. In a child, a general anæsthetic may be needed. To evacuate an insect from the ear it is best to flood the meatus with liquid paraffin.

Foreign body in the nose.

The foreign body is usually found in the inferior meatus. A small Eustachian catheter should be passed backwards above it with its beak downwards. It is then tilted down and withdrawn when the foreign body is usually brought out with it. Should more manipulation be needed, a general anæsthetic may have to be given as the patient is usually a child who will resist local anæsthesia as much as the manipulation itself. Epistaxis (*page 123*) may have to be controlled.

PARAPHIMOSIS

The earlier an attempt is made to draw forwards the œdematous foreskin, the more likely it is to be successful. The distal portion of the penis should be wrapped in a compress of cotton wool



FIG. 66

Method of attempting to reduce a paraphimosis.
(*Pye's Surgical Handicraft.*)

soaked in 0.1 per cent. solution of adrenaline hydrochloride and grasped in the hand for several minutes. The penis is then held as shown in Fig. 66 and the prepuce restored to its normal position. If this manœuvre fails a little hyaluronidase (*page 654*)

(1,500 international units in 5 ml. of 1 per cent. procaine) should be injected at 3 and 9 o'clock positions. This causes reduction of swelling in 10 minutes or so and allows easy reduction.

TRISMUS

The urgency of trismus depends upon the fact that tetanus may be the cause, particularly if it is painless. If this is suspected give 200,000 units of tetanus anti-toxin after taking the usual precautions (*page 579*). Trismus of a few days' duration may set up an intense stomatitis. The danger then is that the trismus may be attributed to the foul mouth and not to tetanus and so urgent treatment may be delayed.

TETANUS

This is a medical emergency with surgical aspects in that the causal wound (often slight) may be opened up (preferably when very recent) and treated with hydrogen peroxide. The "wound" is not uncommonly the result of self-administration of drugs in addicts. X-ray examination for a foreign body may be helpful. Don't assume that the first scratch seen is the causal one. The incubation period of tetanus (as judged from patients with a single injury) is 7 to 13 days (mean 10 days) and so the connection between stiffness and injury may be overlooked and treatment delayed. Stiffness may be attributed to other causes (*see Trismus page 481*) but once tetanus is diagnosed treatment must be prompt. Tetanus is unlikely to be the cause of symptoms if the patient has received active immunisation with tetanus toxoid during the previous five years. The prognosis can generally be gauged by the interval between the first stiffness and the first spasm. If over 48 hours the outlook is good.

Treatment.—All cases should receive 200,000 units of tetanus anti-toxin intravenously after taking the usual precautions (*page 579*).

Cases without severe spasm.

Adequate sedation and feeding must be maintained. Sodium phenobarbitone 0.1 G. (gr. 1½) by injection or mouth every six hours may be enough but any suggestion of spasm calls for stronger measures such as Bromethol B.P. (Avertin). This should be given rectally in doses of 0.075 to 0.1 ml. per Kg. body weight

($\frac{1}{2}$ to $\frac{2}{3}$ m. per pound) every six to eight hours (*see page 102*). Alternatively paraldehyde may be used (*see page 597*).

Cases with severe spasm.

These patients should be treated where supervision by a skilled anaesthetist is available and I am indebted to Dr. J. Ablett of Leeds for the following details.

When spasms are more severe complete muscular relaxation is advised* as it can be effective without causing respiratory paralysis. It should not be given orally as it makes swallowing difficult but by oesophageal tube, initially in single doses of 1.5 G. with a maintenance dose of between 1.25 and 2.0 G. per hour. It can be given intravenously in a 1.0 to 2.0 per cent. solution but intermittent undiluted doses must be avoided as they are liable to cause hæmolysis.

When spasms are more severe complete muscular relaxation should be produced by succinylcholine given as a 0.2 per cent. solution intravenously in saline. Other relaxant drugs (d-tubocurarine, flaxedil and laudolissen) may be used.

TRACHEOTOMY AND ARTIFICIAL RESPIRATION.—The indications for tracheotomy are

- (1) recurring cyanosis in patients under sedation,
- (2) chest infections from inhaled vomit,
- (3) laryngeal œdema,
- (4) full paralysis necessitating artificial respiration.

A careful tracheotomy should be performed under thiopentone and succinylcholine anaesthesia after passage of a cuffed endotracheal tube.

Artificial respiration with a mixture of nitrous oxide, oxygen and nitrogen should be maintained by intermittent bag compression by some form of breathing machine (*page 559*) until the infection has subsided. If air is delivered under positive pressure as by a Beaver pulmoflator efficient humidification is essential.

GENERAL MEASURES.—A high calorie diet is given by oesophageal tube or gastrostoma. The position in bed should be changed two hourly following chest auscultation, tracheal suction and physiotherapy. Daily investigations should include chest X-ray, blood count and estimation of blood electrolytes as well as fluid balance checks. Saline may be given into the femoral vein by a plastic tube.

When spasms no longer appear on ceasing the flow of relaxant drug the anæsthetic should be stopped and the patient allowed to breathe air through the tracheotomy tube. The atmosphere should be warm and moist to avoid drying up of secretions.

ANTHRAX

This is a rare emergency but should be thought of if a worker in the bone and hide trades has vesicles on the hands or face or any carbunculoid condition with a black slough, purulent and bloody discharge and brawny œdema. Laboratory workers are occasionally infected and should remember that staining does not kill anthrax spores.

If anthrax is suspected, material from a vesicle or slough should be sent for examination but treatment should not wait on the result. The part should be immobilised and the lesion painted with 1 per cent. gentian violet. Tetracycline (*page 607*) is the drug of choice but penicillin and erythromycin are equally effective. Anti-anthrax serum (*page 619*) 100 ml. daily by intravenous injection causes severe reactions and is best reserved for resistant cases and for pulmonary anthrax (Woolsorter's disease).

THE FIXED WEDDING RING

A not uncommon emergency is caused by a wedding or other ring which cannot be removed and which causes swelling or even ulceration of the finger. Division of the ring by cutters or a Gigli saw is often performed but most women have a sentimental objection to such methods which sacrifice the ring. The following simple method using a piece of string is very effective and should be tried first.

Beginning near the distal interphalangeal joint fine string is wound tightly round the finger up to the ring and the end hooked under it by a curved needle or pushed under by a match stick (*Fig. 67*). The end is then pulled towards the finger tip and as it unwinds the ring comes with it since the string provides a pull at an infinite number of points. Wedding rings come off well but rings with stones require patience and manipulation in the unwinding process.

If this method fails 1,500 international units of hyaluronidase (*see page 654*) in 1 ml. of 1 per cent. procaine should be injected into the finger and massaged a little. Then a length of thin rubber tubing should be wrapped round the finger from the tip up

to and including the ring and left in place for five minutes. This will often reduce the swelling sufficiently to permit the ring to be drawn off or the string method to be used successfully.

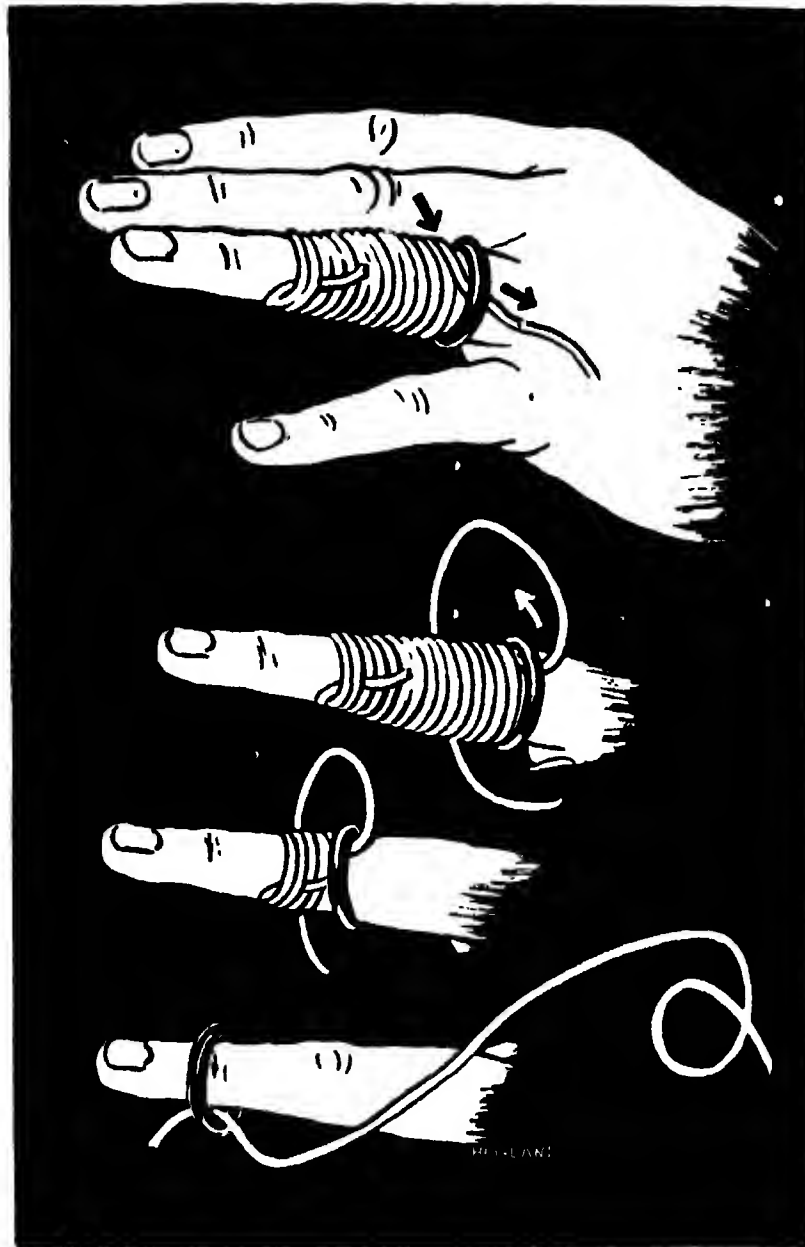


FIG. 67

How to remove a wedding ring.
(Steps in order from above downwards.)

THE BLEEDING TOOTH SOCKET

The bleeding tooth socket not infrequently causes the hospital resident to be summoned urgently. In the usual case the procedure is as follows. Reassure the patient and, if necessary, give

him a sedative. Clear the mouth of loose clots, using cotton wool and physiological saline. Verify the site of bleeding. Make a roll of gauze like a cigar and put it across the socket. Tell the patient to bite on this. A bandage may be used to keep the jaws together. After twenty minutes remove the gauze gently. This is usually all that is necessary as the bleeding was capillary in origin. If bleeding continues there is probably some periosteal separation. Anæsthetise the gum on either side with 2 per cent. procaine and put in a horizontal suture of nylon or silk to draw the gum margins together. Should this fail you should seek help from your senior colleague.

The approach should be rather different in a patient who is a known or suspected bleeder. Tight plugging and suturing should be avoided. A piece of Calgitex gauze soaked in thrombin solution should be put in the cavity and held in position by biting lightly on a larger piece of gauze. Anything more drastic should be avoided, but the patient may be transfused.

ACUTE GOUT

The pain of gout, commonly in the big toe, may be so severe as to occasion an emergency call. Although its mode of action is unknown, there is no doubt that colchicum is the best remedy. Since the tincture is unstable it is preferable to use crystalline colchicine, and many sufferers carry a supply of this as half milligram pills. The initial dose should be 1 mg. (gr. $\frac{1}{64}$) followed by 0.5 mg. at two-hour intervals until pain is relieved or vomiting or diarrhoea result. Four mg. are generally needed. Full dosage may be tolerated if tincture of opium 4 ml. (m 60) is given when vomiting and diarrhoea begin. Intolerance by mouth can be avoided by giving 3 mg. of colchicine in 3 ml. of physiological saline intravenously. Full doses of aspirin may also be given.

A.C.T.H. (Corticotrophin) has a small place in the treatment of the acute attack in those rare cases completely resistant to colchicine. The dose should be 100 mg. a day in four six-hourly doses of 25 mg. intramuscularly. This dose should be "tailed off" over a few days and colchicine 0.5 mg. given three times a day to prevent a "rebound attack." A.C.T.H. should not be given during a remission as it may precipitate an attack. Phenylbutazone (Butazolidin) 600 mg. a day is said to be effective in acute gout.

DRUG ADDICTION AS AN EMERGENCY

If an addict is deprived of his supply of drugs, urgent symptoms may develop as they did, for example, on the train "City of San Francisco" which became snowbound for several days in January, 1952. The following notes are provided to warn the practitioner what to expect and to advise him what to do.

There is no such person as a "registered drug addict." The Home Office may keep files of known addicts and refrain from taking action when there is evidence that the patient is accepting suitable treatment or medical supervision of the use of the drug. Diagnosis is easy if the patient is a self-confessed addict. Suspected addiction, however, may be difficult to prove. There are no pathognomonic signs, but needle marks and abscess scars in parts of the body accessible to the hands such as the back of the left upper arm (in a right-handed addict) or the front of the elbow (when intravenous injections are used), should raise suspicions. "Drunkenness," mental confusion and emotional changes are characteristic of alcohol and barbiturate intoxication but these changes are absent in the morphine addict.

When it is necessary to be certain whether doubtful symptoms are due to morphine, nalorphine may be used—preferably with the patient's written consent. Give 3 mg. nalorphine subcutaneously. If no abstinence symptoms occur within 20 minutes give 5 mg. more and again after 20 minutes 8 mg. more if necessary. If no abstinence symptoms occur after these doses then the patient has not been getting morphine. Nalorphine symptoms, however—slurred speech, slow respiration and small pupils—will be present.

Withdrawal symptoms are similar in the case of all narcotic drugs of addiction but their severity and timing vary. After a symptomless interval restless sleep comes on. Then there is a phase of yawning, perspiration, gooseflesh and running from the nose and eyes; later twitching, great restlessness and insomnia appear. Cramps come on next and there is often vomiting and diarrhoea. Acute mania may occur. Circulatory failure and death ensue unless morphine is given. Sudden withdrawal of large doses of barbiturates and paraldehyde may precipitate convulsions. Smaller doses cause minor symptoms on withdrawal and there is also a good deal of individual variation. Sometimes the presenting

complaint is of severe pain—an attempt by an undisclosed addict to obtain a dose of morphine.

The approximate time of onset of symptoms after withdrawal is as follows:—

Morphine and Dromoran (Methomorphinan) 16 hours (maximal at 48 hours); Dilaudid (di-hydromorphinone) and Heroin (diamorphine) a few hours (maximal at 12 hours); barbiturates 8 to 12 hours; codeine more than 20 hours; pethidine (meperidine) 3 hours; Physeptone (methadone) 3 to 4 days.

The principles of treatment of drug addiction are that it should be carried out in an institution and that no drugs should be given to the patient for self-administration.* If the patient refuses these conditions the doctor should abandon the case. If the symptoms were urgent, however, he may be obliged to act. But he would be wise to arrange admission to hospital first and, having done this, an initial dose of morphine 16 to 32 mg. (gr. $\frac{1}{4}$ to $\frac{1}{2}$) or Physeptone (methadone) 10 mg. should be given. Mephenesin B.P.C. up to 1 G. every three hours by mouth will help to control cramps and restlessness and will reduce the patient's anxiety. These drugs control the abstinence symptoms associated with the use of other narcotics, but severe barbiturate withdrawal symptoms are best relieved by re-intoxication with 0.5 to 1 G. of pentobarbitone (Nembutal). The patient should not be told what he was given.

Urgent withdrawal symptoms should never be allowed to occur in patients with incurable diseases. Should they arise by accident they should be promptly relieved by giving the appropriate drug.

If an addict urgently needs an operation it is best to provide him with his drug until it is all over. If he is a cured addict only the *normal* pre-operative doses of narcotic drugs should be used and the patient should be watched for relapse.

Cocaine and Hashish.

Of the three characteristics of addiction (habituation, tolerance and psychological or physical dependence) only habituation is produced by cocaine and hashish (marihuana). Hence acute withdrawal symptoms do not occur but overdosage may occasion an urgent call.

* In addition to mental hospitals there are certain institutions which accept drug addicts (and alcoholics) for treatment. A detailed list is published by the Church of England Temperance Society, 4 Palace Gate, Kensington W.8. Tel. WEStern 8891.

Cocaine over-dosage shows by sweating, tachycardia, tremor, dilated pupils and an unblinking stare, and the patient has auditory and visual hallucinations. •Intoxication with marihuana causes a sleepy appearance with injected conjunctivæ, giggling and silly behaviour, and a strong characteristic odour in the breath.

Treatment of both these states is on general lines (*see* Chapter II). The morphine antidote N-allylnormorphine (Lethidrone) (*see* page 14) is without effect in cases of overdose with cocaine and hashish.



FIG. 68
Frostbite.

FROSTBITE

This only occurs when the air temperature is well below zero. Unless precautions are taken it can easily occur during flight as the temperature falls 2°C . for every 1,000 feet above sea level until a temperature of minus 55°C . is reached, when it remains constant. The nose, ears and hands are the common sites affected (Fig. 68). An early sign is a hard white patch on the skin detected in "making a face." Humidity is an important factor. At minus 35°C . contact of the skin with a metal surface in the presence of

moisture produces instantaneous frost-bite of a severity comparable with that of a second degree burn of similar extent. Frostbite has occurred when a little liquid N_2O has remained in an open frosted cylinder and has rapidly expanded and cooled when the cylinder was inverted in handling.

PREVENTION.—In cold climates adequate protective clothing and gloves should always be worn. Beards and moustaches should be kept short. In the air prevention depends on efficient cabin heating or upon electrically heated clothing and oxygen administration. The face can be protected by lanoline and the lips by colourless lipstick, which lower the freezing point of the skin. Metal studs and fittings must not be allowed to touch the skin.

Treatment.—The frost-bitten part should be protected by cotton wool and kept at cool room temperature. No attempt should be made to hasten recovery of local warmth and sensation. If frostbite occurs when in the air oxygen and hot drinks should be given and the frost-bitten part kept still. If lividity and induration persist the patient should be transferred to hospital. There is some evidence that anti-coagulant therapy (*see page 595*) may forestall gangrene.

C. ALLAN BIRCH.

CHAPTER XXVII

Medico-legal and other Non-clinical Emergencies

ALMOST all forms of treatment and investigation we use may, on occasion, be harmful to the patient and so lead to legal action. Every practitioner should, therefore, belong to a defence society (preferably the same one as his partner or chief) and report to it *at once* when involved in a situation likely to lead to a claim for damages. At the same time immediate and energetic steps should be taken to reverse the consequences of any accident. Special medico-legal emergencies arising on board ship are described on *page 382* and the question of responsibility for advice given by wireless in an emergency is dealt with on *page 360*.

NEGLIGENCE

Since a charge of negligence may follow the doctor's attention to his patient, a clear statement of what negligence means is desirable. In law, negligence is judged by the standard of prudence of an ordinary reasonable man, but a person who undertakes something requiring special knowledge or skill is negligent if, by reason of his not possessing that knowledge or skill, he bungles, although he does his best. The negligence does not consist in the lack of skill, but in undertaking the work without skill. The physician must exercise such care as a normally skilful member of his profession may reasonably be expected to exercise. This principle applies even though the doctor undertakes to do the work gratuitously. Extraordinary skill, however, is not required of anyone and erroneous judgment in a difficult case does not constitute negligence.

Contributory negligence.

In some cases a patient may, by his own negligence, contribute to his injury and formerly a plaintiff's claim was defeated if the defendant proved contributory negligence. The law has now been amended by the Law Reform (Contributory

Negligence) Act, 1945, which makes liability in cases of contributory negligence apportionable between plaintiff and defendant. Where any person suffers damage as a result, partly of his own fault and partly of the fault of another, a claim in respect of that damage *shall not be defeated* by reason of the fault of the person suffering the damage, but the damages in respect thereof shall be reduced as the Court thinks just, having regard to the claimant's share in the responsibility for the damage.

Even so it is wise to keep notes describing the exact sequence of events, since although doctors are no longer likely to *defeat* a plaintiff's case against them, by establishing contributory negligence, they may secure *mitigation* of damages.

Should the patient refuse to obey instructions in an emergency following a medical accident, the practitioner would be wise to cease treatment, and thus, by establishing contributory negligence, weaken the patient's claim against him. But he would be well advised to give the patient full warning of his intention and of his reasons for it, and also to give him reasonable opportunity to follow his instructions. He should, in fact, endeavour to provide what the Court would regard as clear and ample evidence that he had done everything that an ordinary and reasonably skilful practitioner would be expected to do.

PERMISSION TO ACT IN AN EMERGENCY

The patient should always be consulted and permission obtained before carrying out any procedure other than what is needed in the most sudden and dire emergency. Consent must not be assumed just because the patient arrives at hospital. If consent cannot be obtained, no more should be done than is necessary to deal with the emergency. For any operative procedure or anæsthetic and for some examinations (*see page 498*) consent, preferably written, should be obtained after explanation of what it is proposed to do *in terms which the patient can understand*. Consent must be full, free and valid in all respects. Though written permission is not essential it provides better evidence, in any subsequent proceedings, that consent was given, since witnesses may die and memories fade.

In the case of a child the parents must be consulted and if a minor has passed the age of 16 or has reached a stage of mental and physical development sufficient to enable him to appreciate

what is proposed to be done, his or her consent should be added to that of the parent, guardian or person *in loco parentis*. If the parents cannot be consulted in time, the authority of the headmaster or whoever stands *in loco parentis* should be sought. A full statement of what has been done should be communicated to the parents as soon as possible. It should be noted that a friend, however intimate, has no authority to give permission.

EMERGENCY TREATMENT BY LAY PERSONS

A lay person should render such emergency treatment as he thinks best and would be deemed culpable if he did nothing. Occasionally injection of morphine for painful crises of incurable disease has been delegated to relatives. Although it would be difficult to devise safeguards to cover all possible legal consequences of this procedure, the doctor could arrange it at his discretion. He should make sure that the relative is competent to inject morphine without supervision. Supplies of drugs left with relatives should be small.

DYING DEPOSITIONS AND DECLARATIONS

Although there is no legal obligation on a doctor to take down the statement of a dying patient, he is usually the best person to do this in an emergency. Two situations must be clearly distinguished:—

(1) When the *doctor* thinks the patient is unlikely to recover but the patient is unaware of the imminence of death a **deposition** should be taken. A magistrate should be summoned and it will be his responsibility to see that legal requirements are complied with, *e.g.*, that an accused person or his legal representative should have the right to cross-examine. Such a deposition, properly taken, is admissible in evidence.

(2) When the *patient* is in settled hopeless expectation of death and makes a statement this is a **dying declaration**. It should be written down together with any questions used to elicit full information, and their answers. A phrase such as "Having the fear of death before me, and being without hope of recovery" must be included. The presence of a magistrate is unnecessary. The declaration should be signed by the dying patient (if possible), and the person who writes it down. Witnesses are not necessary, but are desirable. This type of declaration is limited to charges of

homicide and is not admissible as evidence if referring to homicide other than that of the declarant.

HOW TO BEQUEATH A BODY FOR DISSECTION

This is hardly an emergency but I have known information to be sought urgently on behalf of a patient who intended his body to go to a medical school for the benefit of medical education but took no steps until he knew he was soon to die. As the procedure is little known it is described here.

It is not possible for a person to bequeath his body in a legal sense for in law there is no property in a body. The legal right to dispose of a body rests with the person (next of kin, executor or other person who has custody of the body). Even he cannot send the body for dissection if a near relative objects. A person wishing to bequeath his body for dissection should, therefore, take the following steps :—

1. Make sure that near relatives do not object.
2. Make a written statement of his intention preferably witnessed.
3. Apply for the necessary forms (a special medical certificate and a bequest form to be signed by the next of kin or executor) to H.M. Inspector of Anatomy, Anatomy Office, Ministry of Health, Savile Row, London W.1. These forms should be sent immediately after death. An ordinary death certificate is also needed for the Registrar who then issues a Certificate for Disposal which must be given to the undertaker who removes the body. Removal cannot take place until 48 hours after death. It is preferable not to bequeath a body to a particular school but to let it go to whichever is convenient. The Professor of Anatomy is responsible for the body until it is ready for burial. The burial certificate is sent to the Inspector of Anatomy who sends it to whoever authorised the removal of the body. All expenses of a simple funeral but not cremation are borne by the medical school. If the body was the subject of a coroners' inquest this would not prevent its disposal to a medical school though if a post-mortem examination had been made the Professor of Anatomy might then judge it unsuitable for his purpose.

EYES BEQUEATHED FOR THERAPEUTIC PURPOSES

If a patient asks how his eyes may be donated for surgical purposes he should be told to sign a statement (preferably wit-

nessed) of his wishes. It need not be in the Will but the executor should be told. Ideally a copy should be sent to the next of kin, the executor of the will, the family doctor and the hospital (*see page 649*). If the request is oral the person to whom it is made should record it in writing before witnesses.

When the doctor attending a dying patient is told of such wishes he should, after obtaining the consent of the relatives or friends, promptly inform the nearest corneal grafting hospital or collecting centre (*see Appendix VI page 649*) of the impending demise of the patient or should do so as soon as possible after death. The hospital will send out someone to collect the eyes or will make arrangements (*e.g.*, with the local branch of the British Red Cross Society) for this to be done if the eyes are needed. Pending removal Chloramphenicol 0.5 per cent. should be instilled into each eye. (The eyes are enucleated under aseptic precautions within, at most, 12 hours after death and transferred immediately into a sterile screw capped bottle containing liquid paraffin. The bottle is put into cracked ice or kept at 4° C. Enucleation is less urgent if the body is kept in the mortuary cool chamber. Steps should be taken to see that there is no obvious disfigurement of the body).

Bottles containing eyes must be sent by road or rail and *never by post*. The following particulars should be supplied:—

Name or number, age and sex of patient.

Place where eyes were removed.

Date, time and cause of death.

Length of time after death eyes were removed.

Details of any ocular abnormality.

Details of any systemic infection.

CRIMINAL ABORTION

The obligation on a doctor or any third party to report a crime depends on whether the crime is a felony or a misdemeanour. Misprision (concealment) only becomes a crime in the case of felonies. The offence of concealment of a felony is almost obsolete, and a prosecution is most unlikely.

If a doctor contemplates giving information to the police about an abortion he should seek expert legal advice, since there is no certainty that he will be protected against subsequent litigation for defamation.

If the patient is dying the doctor may urge her to make a statement but he should not put leading questions. When she dies the Coroner must be informed. If criminal abortion is only suspected and the patient recovers, there is no obligation to make any report.

Especially during wartime it sometimes happens that the returning husband finds his wife ill. When this illness is caused by abortion and it is clear that the husband could not have been responsible for the pregnancy, the doctor should be careful of his replies to the husband's questions. The making of a defamatory statement to a husband about his wife is "publication" and could render the person making it liable to an action for slander. Such an action could not, of course, succeed if the statement giving rise to it were true, but if the doctor is not sure of his facts he should not say anything and would be wise to let the wife tell the husband herself.

WHAT TO DO IN A CASE OF ALLEGED RAPE

Alleged rape is an emergency situation since conclusive evidence of it soon disappears. Spermatozoa will be probably unrecognisable after 12 hours. Examination should be made at once but never without the person's consent. In the case of a girl under 16 the consent of the parent or guardian is essential. It is best to exclude all relatives and friends and then a detailed history of all the circumstances should be taken. A general examination for scratches and bruises should follow. The woman should then be thoroughly examined in the lithotomy position in a good light. Take charge of underclothing for subsequent examination for seminal stains. Note and take a specimen of any matted pubic hair. Look for bleeding and for bruising of the vulva and tearing of the hymen. Note the condition of the vagina. Using a swab stick moistened with physiological saline make thin smears from the vagina on several glass slides.

If requested to examine a male accused of rape only do it with his full consent (preferably written and witnessed). Tell him of his right to refuse, look for scratches and bruises and examine the penis for recent turgescence and tears of the frenum. Make smears from any meatal moisture.

DEATH-BED WILLS

We have to distinguish between witnessing and helping to make a death-bed will.

Anyone who understands what witnessing a signature means may witness a will. It is not necessary that the witness should know that the document is a will. He or she need not be over 21 years of age. A minor may witness a signature provided he or she has reached such a stage of education as to appreciate fully what is signified by the action of witnessing a signature. The witness is only "attesting" the signature, and is not concerned with the patient's mental state or how the patient disposes of his property. The doctor who attested a will may, of course, be called as an expert witness and asked if he thought the patient was sane when he signed his will.

The practitioner should only help the patient to *make* his will in an emergency, and when a solicitor cannot be found. He must be satisfied that the patient is of "sound disposing mind." The form of words to be used is set out below.

In most instances marriage automatically revokes a will. All names and addresses of possible devisees and legatees, and descriptions of bequests should be full and accurate and any possibly ambiguous words such as "money" should be avoided. If illegitimate children are to benefit they must be named.

Reference to sums of money should be followed by the words "free of duty"; and the residue of the testator's property should be described as "all the remainder of my estate, real and personal."

The testator should sign the will if possible, or make a mark. If he is quite unable to do either, then someone may sign in his place but the testator should, if possible, acknowledge in some way as by nodding his head, that he understands what is being done. If a third person does sign on the testator's behalf, he *must* sign in the presence of the testator, and the testator must be shown to have seen the signature. Two or more witnesses must sign the will *in the presence of the testator* (this is essential) and preferably in the presence of each other, and nothing more must be added. If a special will form is not available the will can be made on a sheet of paper as follows:—

(Full names and addresses must be given in each case and the will should be in ink or typewritten. Should there be more than one sheet each must be signed and witnessed).

This is the last Will and Testament of Me, A. B., of in the County of I revoke all former Wills by me made. I appoint to be the Executor(s) of this my Will.

(Here follows all bequests and instructions. So long as the intention of the testator is clear it makes no difference what form of words is used. In an emergency a person will generally wish to leave everything to one person such as the wife or husband and it is sufficient then simply to say "I hereby bequest and devise all my real and personal property to X absolutely").

In witness whereof I have hereunto set my hand this day of one thousand nine hundred and

Signed and acknowledged by the said A. B., the testator, as and for his (her) last Will in the presence of us, both present at the same time, who, at his (her) request, in his (her) presence, and in the presence of each other have hereunto subscribed our names as witnesses *(leave no space)*.

(Testator's signature).

(Witness's signature, address and occupation).

(Witness's signature, address and occupation).

Witnesses must not be executors or persons who benefit under the Will. A person under 21 years cannot legally make a Will unless he is a soldier or sailor actually on service, and in such cases witnesses are not absolutely necessary.

If the Testator is unable to write, the following clause should be added and he should make a mark.

Signed by the said A. B., the Testator, the contents having been first read over and explained to him, he being unable to write, and having affixed his mark hereto, in the presence of us, who at the same time, at his request, in his presence and in the presence of each other have hereunto subscribed our names as witnesses.

Mark of ... A.B.

(Witness's signature, address and occupation).

(Witness's signature, address and occupation).

POWER OF ATTORNEY

If a doctor sends his patient to a mental hospital, he may be asked what happens to the patient's money. He should advise the relatives to consult a solicitor, but it is helpful to be able to say what the procedure will be.

If the patient is not certified a power of attorney is adequate, provided that the patient is capable of understanding it. This is simply a formal appointment of an agent by a deed. This is usually drawn up by a solicitor and signed by the patient. It usually runs:—

“ Know all men by these presents that I, A.B., of hereby appoint C. D. my true and lawful attorney in my name, or otherwise, and on my behalf to do and execute the following acts and deeds:— (Then follows a description of what the agent is authorised to do). In witness, etc., etc.”

If the patient is certified and the estate is of any size, an application should be made by the patient's solicitor to the Master in Lunacy, The Law Courts, W.G.2., for the appointment of a receiver. In Scotland relatives or, failing relatives, any parties interested or the incapax himself, may petition the Court for the appointment of a Curator Bonis.

THE FAILED SUICIDE

Attempted suicide amounts to a misdemeanour only, and there is therefore no legal obligation upon the doctor to report the matter to the police. He should do this only in those cases which are likely to end fatally, or for which adequate arrangements such as care by relatives cannot be made. In all cases the doctor should do what he thinks best. If he does not think that a prosecution would be in the patient's interest he should not report the case. If the patient refuses hospital treatment and yet is not certifiable the wisest course would be to seek admission through the mental welfare officer to a mental observation ward.

EMERGENCY EXAMINATION AT POLICE REQUEST

An apprehended person can only be examined and the findings disclosed with his consent. The police have no power to order examinations. Prison authorities, however, may order the examination of a prisoner without his consent.

WHAT SHOULD THE DOCTOR SAY IN AN EMERGENCY?

A doctor may be called on suddenly by the police and asked for evidence which might help in the arrest of a criminal. Has he seen a patient with a cut hand or can he lead the police to a young woman who has just had an abortion? In a similar way information may be sought by an employer about an employee and the circumstances may appear to justify an urgent request.

A doctor should never be stampeded by the emergency atmosphere and "official" environment into saying anything he would later regret. If he does speak, however, and tells the truth, he need not fear prosecution for he will be legally correct. But he may be ethically wrong and on this conflict between law and ethics he must make his own decision. If he can consult the person about whom information is sought and get permission to disclose the facts he is clearly in the right. In other circumstances he would be wise to regard his knowledge as confidential and only to disclose it if refusal to do so might be contrary to the public interest or might render him liable to be charged as an accessory. There is no statutory obligation which requires a doctor to make a statement when asked to do so by the police.

ALCOHOLIC INTOXICATION

A situation with urgent aspects arises when a doctor is called upon to examine an alleged drunk in charge of the police. The evidence is fleeting and so examination should be made promptly and recorded at the time. If you are called on behalf of the police tell the patient that he has a right to have his own doctor's opinion. Do not examine him without his consent (preferably obtained before witnesses) for without this the examination would be technically an assault. If he is unconscious or inaccessible there is no objection to examining him on the ground that he cannot consent, though it would be unwise to take a specimen of blood or to use a needle for a similar purpose. Consent to any reasonable examination may be presumed. Always make the examination in the presence of a police officer. For the benefit of the doctor the following headings are given as a guide to ensure that important points which may be needed in court are not overlooked.

HISTORY.

Name of person examined.

Date and all relevant times.

Patient's story including details of food and drink.

Past medical history (recent illness, drugs, injury).

EXAMINATION.

Ask the patient to write his name and address.

General demeanour (hilarious, abusive, truculent, etc.).

Insight into nature of the situation.

Smell of breath.

Evidence of vomiting.

Evidence of head injury.

Condition of eyes (pupils, ocular movements).

Condition of face (flushed, pale, cyanosed, sweating).

There should follow a general examination including B.P., pulse, urine.

SPECIAL TESTS OF CO-ORDINATION.

Ask the patient to read a newspaper and use a telephone directory.

Finger to nose test.

Walking on a chalked line.

Speech.

Spelling test.

Counting money.

After the examination the doctor should tell the patient his opinion. The private doctor who examines the patient may consult with the doctor called by the police as he thinks fit but as he is making the examination at the patient's request he should not discuss the matter with police officers. If invited to write something in the police station's book he should make a non-committal statement such as "I have examined A. B. on. . . ." Should the doctor have prior knowledge of the patient which might influence his opinion he would be wise to refuse to make the examination.

OPINION.—Three questions about the patient should arise in the doctor's mind:—

(1) Is he ill?

i.e. could his present state be brought on by illness such as G.P.I. or by drugs or insulin?

- (2) Is he "drunk"? *i.e.* is alcohol responsible for his state?
- (3) How "drunk" is he? *i.e.* is his ability to perform some special act, such as driving a car, impaired?

Since individuals react differently to alcohol according to their make-up and state of health, it is difficult to draw a sharp line between the sober and the "drunk." It is best not to say whether a person is "drunk" but merely whether he is under the influence of alcohol to such an extent as to be incapable of performing the act in question. It must not be forgotten that the drink may have contained other toxic substances, *e.g.*, methylated spirit (*page 12*). Absinthe (banned in U.S.A.) contains extract of wormwood, an active narcotic poison which may cause convulsions.

Concussed or drunk? is often the problem if there has been an accident. The car driver may have been drunk before he was concussed or he may smell of drink though sober but concussed because of liquid "first aid." The history from onlookers may help. The patient, if concussed, has no clear recollection of what happened, but the alcoholic, though confused at first, becomes clearer on questioning.

In a person under the influence of alcohol the temperature is subnormal; the pulse rapid and bounding (in shock following an accident it is rapid but thin and thready). The pupils are dilated and sluggish. (In coma they become small but will often dilate when the patient is shaken (Macewen's sign)). The tongue is dry. The patient's breath smells of alcohol, and his speech is slurred. The patient tends to gesticulate wildly and breaks down emotionally during conversation. Hiccup, salivation, drowsiness and confusion are also significant signs.

In assessing the results of special tests it must be remembered that alcohol may dangerously impair judgment before it spoils the performance of these tests and occasionally it seems to improve the response to one of them. Several tests should therefore be used and allowance made for the fact that sober but nervous patients may perform them badly. Too much reliance should not be placed on any of the tests and particularly on the more acrobatic ones.

Blood alcohol.

A percentage concentration of absolute alcohol in the blood of 150 mg. per 100 ml. is generally taken as the level of intoxication. A level of 400 mg. per 100 ml. would probably cause coma. Estimations may be very useful though they are not current practice in Britain. Five ml. of blood should be taken and oxalated, though it is unwise to do this if the patient is unconscious. A note should be made of the amount and type of beverage, and time when it was taken. Laboratory tests of alcoholism are based on the fact that alcohol becomes equally distributed in all body fluids. Hence, since the alcohol content of the urine corresponds to the average blood concentration during the time of its secretion, it is wise to save a specimen of urine. In the case of violent death valuable evidence as to whether the deceased was drunk when he died can be obtained from estimating the alcohol content of blood and urine if these are taken within 48 hours of death and refrigerated.

Treatment.—This is not usually an urgent matter for the patient will recover in time though if methylated spirit has been drunk special measures are indicated (*page 12*). If there is doubt about head injury watch the patient for, as Osler said “Better admit a patient to hospital dead drunk than turn him away to be discharged from the jail dead sober a little later.” An alcoholic who genuinely desires to give up drinking can be helped by Alcoholics Anonymous (BM/AAC London W.C.1. Tel. FLAXman 9669) who will put him in touch with persons in many parts of the country and the world who will stand by him during the emergency period of urgent craving. A list of special institutions accepting alcoholics is kept by the Church of England Temperance Society (*page 187*).

EMERGENCY ASPECTS OF DEATH**Signs of death.**

While the fact of death is usually easily recognised there are cases where a decision is difficult and has certain emergency aspects such as whether to summon special aid and appliances.

In drowning and electrocution we should not too readily assume that biological death has occurred. Artificial respiration should go on for two hours after all signs of life have ceased or until an E.C.G. can be done and shows, over a period of five

minutes, no evidence of cardiac activity. In other cases, *e.g.*, sudden coronary occlusion, it is possible to decide earlier that the patient has passed *flammanitia mœnia mundi* because there is evidence (silence on auscultation) that the circulation has ceased. Even so, death cannot be considered as certain, in these cases, unless the heart has stopped beating for at least five minutes. To be sure by palpation that the pulse is absent or that the nail bed changes colour on the application and release of pressure is difficult in a doubtful case. Fragmentation of the column of blood in the retinal vessels ("cattle trucking") is a more useful sign.

The time of death.

As the doctor may later be asked how long a patient had been dead when seen, he should collect at the time the evidence (rectal temperature) on which to base his opinion. Since the rate of fall of body temperature after death is approximately 1.5°F. per hour in room temperature between 50°F. and 70°F. the number of hours since death is given by the formula

$$\frac{98.4 - \text{rectal temperature}}{1.5}$$

The result should be interpreted with caution.

Rigor mortis should be noted. A rough time-table is that it starts on the face in 6 hours and reaches the hands and feet at 12 hours; it stays for 12 hours and takes 12 hours to pass off.

DEATH CERTIFICATE

The issue of a death certificate is not an emergency procedure though it often takes place in an atmosphere of stress. The young doctor may not know where to turn for information or exactly how to do it and so the following account of the procedure is given.

By the Deaths Registration Act, 1953 all deaths must be certified if a doctor has attended the patient during his last illness irrespective of whether the doctor or someone else has reported the case to the coroner. The doctor who certifies must be the registered (or provisionally registered) medical practitioner who has been in attendance during the deceased's last illness. He must also have seen the deceased within fourteen days before death or his body after death (but not necessarily both). Unless it is for cremation there is no legal obligation on the doctor to see the body after death but he must be satisfied, *e.g.*, from the

ward sister's report, that the patient is dead. The section " ^{seen}not seen after death by me " on the certificate is included to help the registrar to decide whether to report the death to the coroner.

The procedure should therefore be as follows: Carefully observe the rules of your hospital about the time and place for signing death certificates.

Sign the certificate (for placing in the envelope addressed to the registrar); the Notice to Informant (for handing to the informant) and the counterfoil (for retention). The certificate must be sent to the registrar "forthwith."

Don't keep the relatives waiting or delay signing until after an autopsy since this would mean two visits for them—one to give permission and another to get the certificate. Give the certificate at once so that funeral arrangements can proceed. In the case of a cremation certificate, however, it is generally best to complete this after autopsy since the cause has to be certified " definitely " and not just to the best of your knowledge and belief.

If you do not know the cause put down " not known " or " uncertain." In such a case and also if the death was one of those which the registrar is bound by law to report to the coroner (*see page 505*) it will save time if you report the case yourself. You should then initial the back of the certificate at A.

If there is an autopsy, initial space B on the back of the certificate and correct the diagnosis later, if need be, when asked.

If you see a patient for the first time who is moribund and who dies in the receiving room you are not qualified to give a death certificate since it has been held that one attendance is not sufficient to justify your stating that you attended during the last illness. You should ring up the home doctor and if he cannot or will not give a certificate then you should inform the coroner.

Scottish death certificates differ in several ways from those in use in England and Wales. The causes of death are stated in the reverse order from that used in England and the doctor is not required to say whether they were confirmed by post mortem or whether he has seen the body after death. There are no provisions for initialling the back of the certificate; there is no Notice to Informant; certain particulars about pregnancy are required and the certificate must be delivered to the registrar within seven days.

NOTIFICATION OF THE CORONER

The Registrar of Deaths is the chief person whose statutory duty it is to report to the coroner (or in Scotland the Procurator Fiscal) deaths which seem to him to come within the coroner's jurisdiction. There is no similar statutory duty laid on a doctor as such though this duty may apply to him in his capacity as head of an institution such as a mental hospital. It may be that the coroner has a social or moral claim on the doctor as on anyone else to report an unnatural death to him but as such a claim would have no statutory basis and as in any case the test of enforcement could not be applied, no claim of this sort is exercised. The doctor and, indeed, anyone else should be careful that he could not, by any intended act or omission, be held to be obstructing the coroner in his duties. A system of cordial co-operation has grown up whereby "coroner's cases" are reported direct to the coroner by the doctor and this is very convenient to all concerned. If you are in doubt as to whether the coroner should be informed, consult your chief or the records officer or have a word with the coroner's officer himself.

Deaths which it is the registrar's statutory duty to report to the coroner are:—

1. those where a registered medical practitioner was not in attendance,
2. those where no certificate can be given,
3. those where the deceased was seen neither after death nor within fourteen days before death,
4. those where the cause of death is unknown (i.e., where there is an "uncertain" certificate),
5. those where death was unnatural, due to accident, violence or neglect or where it occurred under suspicious circumstances,
6. those where death occurred after an operation,
7. those where death was due to abortion, industrial disease or poisoning.

The list of industrial diseases is modified from time to time and includes—anthrax, chrome ulceration, compressed-air sickness, epitheliomatous ulceration, toxic anæmia, toxic jaundice, and poisoning by aniline, arsenic, benzene, beryllium, carbon disulphide, lead, manganese, mercury and phosphorus. It would be

wise to include also deaths from alcoholism and drug addiction and any unusual diseases like glanders or those of possible accidental origin like Weil's disease.

Some coroners ask that all deaths within twenty-four hours of admission to hospital should be notified to them, but this a purely private local arrangement.

Post mortem emergencies.

Even in the mortuary, emergency situations may arise. An attendant once raised the alarm because he found blood dripping from the head of a corpse. Investigation showed that bleeding originated in the severed jugular veins following a partial autopsy (brain only).

WHAT TO TELL THE PATIENT WHO HAS A FATAL DISEASE

The doctor is often faced with a very ill patient who asks what is really the matter and whether he will get better. This is really an emergency situation and much harm can be done and mental suffering caused by inexperienced handling of it. It is well to be prepared, therefore, for sudden questions of this kind. Where there is life there is hope and a good rule is never to tell a patient, and particularly a young patient, the hopeless truth for as Sir Frederick Treves advised, "In the face of misfortune it is merciless to blot out hope." Do not consider whether you will be thought clever or not but remember Sir Alfred Fripp's words "If we cannot be clever we can always be kind." The criterion to be satisfied should always be what is best for the patient from the medical point of view. In most cases it is possible to give some fairly plausible and partially true explanation of the symptoms and to offer some hope that in the course of time things will be put right. Often disease and drugs dull the senses and there are few patients who when seriously ill can say with Dr. Johnson "I have prayed that I may render up my soul to God unclouded." The relatives should be told the true position but care should be taken that they do not by word or attitude convey anything but hope to the patient.

There are occasions when a patient who is likely to die soon should be given an opportunity to wind up his affairs but this can usually be done in circumstances other than those of an emergency. When a death bed will (*page 596*) or dying declaration (*page 492*)

is made the patient will know that he is about to die and his plight need not be hidden from him.

ON WHAT TO DO WHEN THERE IS LITTLE TO BE DONE

One is sometimes called urgently by the relatives to the bedside of a patient dying a lingering death from advanced carcinoma. In such patients who have developed a high tolerance for morphine, the following euphoric mixture is useful:—

Cocaine Hydrochloride	...	10 mg. (gr. $\frac{1}{8}$)
Morphine Hydrochloride	...	15 mg. (gr. $\frac{1}{4}$)
Clarified Honey	... 4 ml. (m 60)	
Gin	... 8 ml. (m 120)	
Water	... to 30 ml. (1 fl. oz.)	

To be taken as often as required.

Gin is not obtainable on a National Health Service prescription and Rectified Spirit 8 ml. (m 120) with Tincture of Capsicum 0.03 ml. (m $\frac{1}{2}$) in it should be substituted. Syrup 8 ml. (m 120) is often more easily obtained than Honey 4 ml. (m 60).

EMERGENCY BAPTISM

Any child which shows signs of life is qualified to be baptised even if it is not viable. It is not usual to baptise prematurely delivered products of conception which do not show signs of life. In deciding whether to baptise or not the wishes of the parents should be carried out.

Baptism constitutes reception as a Christian and not as a member of a particular church. It may be administered by anyone, male or female, having the use of reason whether baptised or not and irrespective of religious belief provided he intends to do what the Church does. Two things are necessary, (1) Invocation of the Holy Trinity and (2) the use of water. Lay baptism should be performed only in the case of necessity, but the reality of the necessity is for the person performing it to decide. Even if the urgency is not great the validity of the baptism is not affected provided the proper matter (*i.e.*, water) and form of words are used. Godparents are not necessary for private or emergency baptisms.

The doctor's fingers moistened with water should touch the child's forehead while he says:—"A. . . . B. . . ., I baptise thee in the Name of the Father, and of the Son, and of the Holy Ghost." This simple form is valid for Roman Catholics as for

others, and is not afterwards to be repeated. It is valid and final whether the child lives or dies. A name is not necessary for emergency baptism. If there is any doubt particularly in the mother's mind as to whether baptism has been already performed or whether the child is alive, the baptism should be made conditional by prefacing the above words with "If you can be baptised" The clergyman of the parents' denomination should be informed.

In the case of a moribund nameless foundling, it is for the doctor to decide whether to baptise or not. If he does administer baptism it should be conditional and a surname should be chosen afterwards. A clergyman of the Church of England should be informed unless the circumstances strongly suggest alternative procedures, such as the discovery of the child in a Roman Catholic church. It is well to choose names which would not handicap or embarrass him should he survive. The Local Authority who is his guardian has the right to choose names but will usually accept those given at an emergency baptism. The birth is registered by the Children's Officer of the local authority after application to the Registrar General.

Special points applicable to Roman Catholics.

Roman Catholics require the water to run, and therefore prefer pouring to sprinkling. Other points are expressed in the Code of Canon Law 1918, from which the following translation is made:—

Canon 746, §1. No human being enclosed in the mother's womb may be baptised so long as there is probable hope of administering baptism after birth.

§2. If the head presents and there is danger of death it must be baptised on the head; if born alive it must not be baptised again conditionally.

§3. If other parts present and there is danger of death, the presented part must be baptised conditionally; if born alive baptism must again be administered conditionally.

§4. If a pregnant mother dies, the foetus must be extracted by those whose duty it is, and if certainly alive baptised absolutely; if doubtfully alive baptised conditionally.

§5. A foetus baptised in the womb must be baptised again conditionally after birth.

Canon 747. Care should be taken that every foetus born prematurely, no matter at what stage of growth, shall be baptised absolutely if certainly alive, conditionally if doubtfully alive.

Canon 748. Monstrous and unusual forms should always be baptised at least conditionally; if it is doubtful whether there is more than one individual, one must be baptised absolutely and the others conditionally.

Canon 749. Foundlings must be baptised conditionally, unless after careful investigation their certain baptism has been established.

CHAPTER XXVIII

Practical Procedures

WEIGHTS AND MEASURES

TO conform with modern measurements of doses these are all given in the "Metric" system but in the case of older drugs the approximate Imperial equivalent is also shown. In prescribing, however, it will be found convenient to use the *nearest familiar dose* in the "Metric" system to the dose in the Imperial system rather than the *exact equivalent*. For example, while in the United Kingdom we prescribe morphine gr. $\frac{1}{6}$ or $\frac{1}{4}$ (approximate metric equivalents 11 and 16 mg.) it is more usual in countries using the "Metric" system to prescribe 10 or 20 mg. When giving medicines prescribed in the Imperial system it is best to avoid domestic measures and to use a measuring glass because, although the British Standard specification for a medicinal teaspoon is 60 m or 3.6 ml., the size of the domestic teaspoon varies from 2.4 to 7.0 ml.

The following tables give the approximate "Metric" equivalents of Imperial doses. (See also Preface to Second Edition page vi).

WEIGHTS

Imperial	"Metric"	Imperial	"Metric"
1 oz.	30 G. (grammes)	$\frac{3}{8}$ gr.	24 mg.
75 gr. (grains)	5 G.	$\frac{1}{3}$ gr.	22 mg.
60 gr.	4 G.	$\frac{1}{4}$ gr.	16 mg.
45 gr.	3 G.	$\frac{1}{5}$ gr.	13 mg.
30 gr.	2 G.	$\frac{1}{6}$ gr.	11 mg.
15 gr.	1 G.	$\frac{1}{8}$ gr.	8 mg.
10 gr.	0.65 G.	$\frac{1}{10}$ gr.	6.5 mg.
$7\frac{1}{2}$ gr.	0.5 G.	$\frac{1}{12}$ gr.	5.4 mg.
7 gr.	0.45 G.	$\frac{1}{16}$ gr.	4 mg.
6 gr.	0.4 G.	$\frac{1}{20}$ gr.	3.2 mg.
5 gr.	0.32 G.	$\frac{1}{32}$ gr.	2 mg.
4 gr.	0.25 G.	$\frac{1}{64}$ gr.	1 mg.
3 gr.	0.2 G.	$\frac{1}{100}$ gr.	0.65 mg.
$2\frac{1}{2}$ gr.	0.16 G.	$\frac{1}{120}$ gr.	0.54 mg.
2 gr.	0.13 G.	$\frac{1}{160}$ gr.	0.4 mg.
$1\frac{1}{2}$ gr.	0.1 G.	$\frac{1}{200}$ gr.	0.32 mg.
1 gr.	65 mg. (milligrams)	$\frac{1}{250}$ gr.	0.26 mg.
$\frac{3}{4}$ gr.	50 mg.	$\frac{1}{320}$ gr.	0.2 mg.
$\frac{2}{3}$ gr.	45 mg.	$\frac{1}{640}$ gr.	0.1 mg.
$\frac{1}{2}$ gr.	32 mg.		

LIQUID MEASURES

Imperial	" Metric "	
1 pint (20 fl. oz.)	568	ml. (Millilitres) (Approx. 600 ml.)
12 fluid ounces	340	ml.
8 fluid ounces	227	ml.
6 fluid ounces	170	ml.
4 fluid ounces	114	ml.
3 fluid ounces	85	ml.
2 fluid ounces	57	ml.
1 fluid ounce	28·4	ml. (Approx. 30 ml.)
60 ℥ (minims)	4	ml.
50 ℥	3	ml.
45 ℥	2·7	ml.
30 ℥	1·8	ml.
20 ℥	1·2	ml.
15 ℥	0·9	ml. (Approx. 1 ml.)
10 ℥	0·6	ml.
8 ℥	0·5	ml.
5 ℥	0·3	ml.
3 ℥	0·18	ml.
2 ℥	0·12	ml.
1 ℥	0·06	ml.

MILLIEQUIVALENTS

Chemical substances in body fluid react together not by their absolute weights but by their equivalent weights. (The equivalent weight of an element is that weight which will combine with or take the place of 8 parts by weight of oxygen. For univalent elements the equivalent weight is the same as the atomic weight; for divalent elements it is half the atomic weight and so on). It is convenient to express in terms of milliequivalents substances, such as acids and bases, which react together. Acid radicles are chloride, bicarbonate (expressed in terms of CO_2), phosphate, sulphate, organic acids and proteins. Basic radicles are sodium, potassium, calcium and magnesium. A milliequivalent of any substance is the amount contained in 1 ml. of its normal solution. (A normal solution contains the equivalent weight of a substance in grammes per litre. The term "normal saline" however is physiological or isotonic saline and is only about one-sixth the strength of chemically normal saline). 1 milliequivalent of sodium is 23 mg. and so on.

The term milliequivalent per litre (mEq/litre) means the thousandth part of the equivalent weight of the substance in grammes per litre. The simplest way of converting mg. per 100 ml. into mEq/litre is to express the substance as mg. per litre and divide by its equivalent weight (=atomic weight ÷ valency), i.e.,

$$\text{mEq/litre} = \frac{\text{mg. per 100 ml.} \times 10}{\text{equivalent weight.}}$$

Conversely, mEq/litre can be converted into mg. per 100 ml. by multiplying mEq/litre by the equivalent weight and dividing by 10,

$$\text{i.e., mg. per 100 ml.} = \frac{\text{mEq/litre} \times \text{equivalent weight.}}{10}$$

The cases of bicarbonate and proteins require special explanation.

Bicarbonate.—We measure the plasma bicarbonate by the amount of CO_2 which it will yield—normally about 60 ml. per 100 ml. of plasma or 600 ml. per litre. Now the molecular weight in grammes of any gas occupies 22.4 litres at normal temperature and pressure. In the case of CO_2 the molecular weight is also the equivalent weight. If 600 is divided by 22.4 we get 27 which is the figure for the mEq/litre of CO_2 derived from bicarbonate.

Proteins.—Proteins act as weak acids and are expressed in terms of the amount of base they neutralise. The average equivalent weight of proteins is that part of their molecular weight which will neutralise 1 equivalent of NaOH. When the concentration of protein in mg. per litre is divided by this we obtain a figure for the milliequivalents of protein. It is more simple to use the formula:

$$\text{Protein in G. per 100 ml.} \times 2.43 = \text{mEq/litre.}$$

Blood plasma has normally about 7 G. protein per 100 ml. and thus has the power of binding about 16 mEq/litre of base.

Equivalent weights of common substances are:—sodium 23; potassium 40; calcium 20; chlorine 35.5; sodium chloride 58.5; bicarbonate 22.4. (The latter is not strictly the equivalent weight, but as explained above, is used in the same way).

IDENTIFICATION OF TABLETS

Many doctors prescribe tablets which they rarely see and with whose appearance they are unfamiliar. Loose tablets have sometimes to be identified in an emergency and so a study of what various tablets look like is worth while. Although the addition of

colouring to B.P. tablets other than those specified is not official and pharmacists generally disapprove of recognising tablets by colour, the doctor, for his own convenience, may consider it advisable to select for his bag brands of tablets of distinctive marking and colour so as to lessen any chance of confusion in an emergency. Some tablets, such as Gantrisin, Tromexan, Stovarsol and Disprin have their full name impressed on them. The distinctive markings (usually an abbreviation of the chemical name or the maker's name) of some others are as follows:—Sulphamezathine—SZ; Sulphadiazine—SDZ; Sulphamerazine—SMZ; Mepacrine—Q; Dilaudid (and other Knoll tablets)—K.

THE USE OF THE TELEPHONE IN EMERGENCIES

In order to call the police or an ambulance the telephone should be used in accordance with the instructions issued by the Post Office. With the automatic (dial) telephones of London and certain other places, 999 should be dialled. This does not call the police or ambulance direct but makes a special signal at the telephone exchange. The call is then passed to the appropriate emergency authority by the operator according to the request of the caller. In some areas emergency calls are made by dialling "0" or "01" or by pressing an emergency button.

EXTRACT FROM RULES FOR THE BROADCASTING OF S.O.S. AND SIMILAR MESSAGES

1. For relatives of sick persons.

The British Broadcasting Corporation (Broadcasting House, London, W.1., Telephone LAngham 4468) will broadcast messages requesting relatives to go to a sick person only when the hospital authority or the medical attendant certifies that the patient is *dangerously ill*, and if all other means of communication have failed. In the normal course of events messages will be broadcast only when the full name of the person wanted is available.

NOTE.—When the person sought is known to be on board a ship at sea, a message can only be broadcast if the ship is not equipped with apparatus for the reception of messages by wireless telegraphy. Further, there must be a possibility that the return of the person sought can be hastened by the reception of such a message. This is not considered to be so when the ship

is on its way to a known port. In such cases, enquirers are advised to communicate with the owners or agents of the ship or with the port authorities.

In no case can an S.O.S. be broadcast requesting the attendance of relatives *after death has occurred*.

2. Appeals for special apparatus, foods or medicines.

These appeals will be broadcast only at the request of major hospitals, and after every other means of obtaining the required item has failed.

No message can be broadcast regarding lost animals or property, except where there is real danger to life, as from the theft of dangerous drugs or from escaped wild animals, and then only at the request of the police.*

There is no charge for broadcasting S.O.S. messages.

INFECTIOUS DISEASES IN GENERAL WARDS

The occurrence of an infectious fever in a general ward creates an urgent situation for the doctor in that action and advice are needed at once. The following notes are intended to guide him.

There is no rule of thumb plan about isolation and quarantine. Our aim should be to prevent spread of the disease to susceptible patients, and at the same time, not to interrupt the work of the ward unnecessarily. Surveillance often achieves this object better than quarantine, but the nursing staff should be told what to look for, *e.g.*, slight coryza and rise of temperature. Measles, rubella, chicken-pox and mumps can be regarded as almost inevitable and it is a good thing if healthy children can get over them before they grow up. Elaborate measures to escape them are not to be encouraged except in special cases. Any members of the staff who have not had the disease in question should be carefully watched. It is best to let them leave the children's or maternity ward for an adult ward.

Erysipelas, meningitis and poliomyelitis are of low infectivity and patients can be nursed in general wards, if otherwise convenient, by barrier methods. In order to obtain special treatment and for administrative reasons it may be wiser to transfer them.

In the case of certain diseases the Medical Officer of Health must be notified. In some areas he wishes to know about rubella

*If a doctor loses his bag he should emphasise the fact that it contained dangerous drugs. This will expedite measures to recover it.

also and other diseases may be made notifiable in special circumstances, *e.g.*, chicken-pox during an epidemic of smallpox.

The following is a list of notifiable infectious diseases in England, Wales, Scotland and Northern Ireland:—

Cholera.	Pneumonia, Acute Influenzal.
Diphtheria.	Poliomyelitis, Acute.
Dysentery.	Psittacosis (N. Ireland only).
Encephalitis (Acute) (Scotland	Puerperal Pyrexia.
Infective N. Ireland Leth-	Relapsing Fever.
argica).	Rheumatic Fever (N. Ireland
Enteric (Typhoid and Para-	only).
typhoid) Fever.	Scarlatina or Scarlet Fever.
Erysipelas.	Smallpox.
Gastro-enteritis in children under	Trachoma (N. Ireland only).
two years (N. Ireland only).	Tuberculosis (N. Ireland to
Malaria.	Tuberculosis authority).
Measles (not in Scotland).	Typhus.
Membranous croup.	Undulant Fever (N. Ireland
Meningococcal Infection (Scot-	only).
land and N. Ireland—Cerebro-	Vincent's Angina (N. Ireland
spinal Fever).	only).
Ophthalmia Neonatorum.	Whooping Cough.
Yellow Fever (N. Ireland only).	Food poisoning (or suspected
Pemphigus (N. Ireland only).	„ food poisoning) (not in Scot-
Plague.	land; N. Ireland Bacterial
Pneumonia, Acute Primary.	only).

Measles.—A child who contracts measles should be sent home or to the isolation hospital. If this is impracticable isolate him in a separate room and use barrier nursing techniques. Find out which of the other children have had measles. (This should be stated on the admission sheet but it is as well to verify the evidence). Nothing further need be done about these children. Give the parents of children who are well enough and who have not had measles the option of taking them home, possibly to develop this inevitable disease.

Gamma globulin (obtainable from The Central Public Health Laboratory, Colindale Avenue, London N.W.9. Telephone COLindale 7041) may be used to prevent or modify an attack. It is difficult and expensive to prepare and its use should be confined to children who have some other serious disease or who are “weakly.” If the supply is short restrict its use to children in beds next to the measles case rather than give a smaller dose all round. As the attack rate in infant contacts under six months is low they need not be given gamma globulin if one can be quite sure that the mother has had measles.

Gamma globulin is issued in ampoules containing 250 mg. of protein which should be dissolved in 3 ml. of sterile distilled water. It is given subcutaneously or intramuscularly within two or three days of the rash in the infecting case in doses according to the following table. Immunity conferred by gamma globulin lasts from three to four weeks.

	<i>Under 1 year</i>	<i>Over 1 year and under 3 years</i>	<i>Over 3 years</i>
Prevention . .	3 ml. (250 mg.)	6 ml. (500 mg.)	9 ml. (750 mg.)
Modification .	3 ml. (250 mg.)	3 ml. (250 mg.)	3 ml. (250 mg.)

If gamma globulin is unobtainable convalescent serum (preferably from a single donor) may be used. It should be given from the first to fifth day after exposure in doses of 0.3 ml. per pound (0.6 ml. per Kg. approx.) body weight (for prevention) or 0.1 ml. per pound (0.2 ml. per Kg. approx.) body weight (for modification). The use of pooled adult serum should be abandoned because of the risk of serum hepatitis. If preventive doses are given, admissions and discharges can go on as before.

Mumps.—The primary case should be sent home or to the isolation hospital. Nothing need be done for fourteen days and then, if there are susceptible children left, the ward should be closed for a week. The children should be watched for early symptoms. The likelihood of cases developing after the twenty-first day is very slight. Only children who are definitely known to have had mumps should be admitted during the period of surveillance.

Rubella.—This is not highly infectious and secondary cases are not inevitable. Hence when the primary case has been disposed of, surveillance only should be practised. A child should not be sent home if his mother is in the early stages of pregnancy (*i.e.*, up to the end of the fourth month). If there has been contact the mother should receive 750 mg. of gamma globulin.

Diphtheria.—The patient should be isolated—usually in the infectious diseases hospital. All contacts (patients and staff) should be questioned about immunisation and their throats and noses should be swabbed. Those who are positive should be isolated, and the others should be carefully watched. Un-immunised children and staff with negative swabs may be given passive immunity (which lasts fourteen to twenty-one days) by antitoxin (2,000 to 4,000 units intramuscularly). Active immunisation should also be started.

Scarlet fever.—The case should be sent home, or to the isolation hospital or to a separate room and treated with penicillin. Scarlet fever is usually mild and uncomplicated but it should not be regarded lightly in a surgical or maternity ward. Throat swabs should be taken from the remaining patients and staff and those who show hæmolytic streptococci or who have symptoms should be isolated and given penicillin. This eliminates the streptococci in four or five days and drug resistance does not develop. The ward need not be closed for admissions because of a single case but closure may be necessary for a few days if further cases occur.

Chicken-pox.—The child should be sent home or to an isolation hospital. Chicken-pox is very infectious and as cases are not usually diagnosed in the early stages secondary cases can be expected. A history of chicken-pox should be regarded as doubtful if careful search does not show any pock marks. If susceptible children are left in the ward only patients who give a definite past history of chicken-pox should be admitted for twenty-one days—the quarantine period. An adult with herpes zoster may give rise to chicken-pox in child contacts.

Smallpox.—If this is suspected ring up the Medical Officer of Health and act on his instructions.

Whooping cough.—The case (suspected because of a spasmodic cough and a lymphocytosis or proved by a per-nasal swab), should be sent home or to the isolation hospital. Further cases will not occur until the end of the incubation period—say in seventeen days. Hence admission of children with a definite past history of whooping cough can go on for the first ten days. Children in the ward who have not had whooping cough may be given gamma globulin as a prophylactic in doses double those recommended for measles. Admissions should cease from the tenth to twenty-first

day after the case has been received and the remaining children should be carefully watched.

Sonne dysentery.—The patient should be sent to the isolation hospital or isolated under strict barrier nursing precautions until several consecutive stool cultures are negative. Rectal swabs, or preferably stools, of all contacts should be examined. Members of the staff must report any diarrhoea. Admissions need not be restricted.

	INCUBATION in days	INFECTIVITY	QUARANTINE in days
Chicken-pox	Extremes 10 to 16 Commonest 14	Until all scabs are off	21
Diphtheria	2 to 10	Until two consecutive nose and throat swabs are negative	7
Measles	7 to 14 Normally Koplik's spots on the 9th day and rash on the 12th day	14 days from the appearance of the rash or, in mild attacks, until the end of clinical symptoms	21
Rubella	5 to 21	7 days from the appearance of the rash	21
Mumps	Extremes 10 to 28 Commonest 17	1 week after glands subside	28
Whooping cough	14 to 16	21 days or until all catarrh has gone	14

FIG. 69

Incubation, Infectivity and Quarantine.

EMERGENCY TRAVEL ABROAD

Most invalids can be accepted for air travel, but may be refused, unless accompanied by a doctor or nurse. The cost of this, and possibly that of displacing additional seats to accommodate a stretcher, may deter the traveller. The St. John Ambulance Brigade can provide Air Attendants.* Doctors requiring advice regarding the carriage of invalids should consult the Medical Department of the air line concerned (*see also page 398*). Health certificates are a commoner cause of trouble.

Although most countries are now signatories to the International Sanitary Regulations (1951), of the World Health Organisation, the requirements of certain foreign governments for certificates of immunisation may vary and are apt to be changed. Travellers, particularly to the East, should therefore consult the Traffic Departments of the air line or shipping companies, accredited travel agents, or the Embassy or Consulate of the country concerned. The following notes are provided to help any doctor who is asked to advise on health certificates by someone suddenly called abroad. They apply to all forms of travel whether by land, sea or air.

In addition to the specific requirements of particular countries, all persons going overseas are advised to be effectively inoculated against typhoid and paratyphoid. For typhoid and any other disease, for which there is no international certificate, an ordinary certificate from any doctor is sufficient.

International certificates of vaccination are now only necessary in the case of smallpox, yellow fever and cholera. In the case of vaccination against smallpox, if the first attempt at primary vaccination fails, at least two more attempts with different batches of lymph should be made, and the results recorded (*see page 611*).

Any doctor can vaccinate against smallpox and cholera and certify, but yellow fever inoculation and certification can only be carried out at special inoculation centres (*see page 635*). British European Airways Medical Services at Northolt, Renfrew and London Airport can provide inoculation against smallpox and

* For these apply on Monday to Friday, from 9.30 a.m. to 5.30 p.m. to Officer in charge of Register, St. John Ambulance Brigade Headquarters, 8 Grosvenor Crescent, London, S.W.1. Tel. SLOane 9861, and on Saturday and Sunday, 8 a.m. to 10 p.m. to Officer in charge of Register, St. John House, 15/16 Collingham Gardens, London, S.W.5. Tel. FRObisher 6477.

typhoid but not against yellow fever. Blank forms of the international certificate against smallpox or cholera must be obtained by the traveller from the transport company, or from the Ministry of Health, 23 Savile Row, London, W.1. (Tel. REGent 8411), or the Department of Health for Scotland, St. Andrew's House, Edinburgh, 1 (Tel. WAVErley 7241). It is essential to have the doctor's signature on any international certificate (other than a yellow fever certificate), authenticated by the local Medical Officer of Health. Certificates of inoculation against smallpox and cholera by practitioners resident in Eire will be authenticated if forwarded to the Department of Health, Customs House, Dublin.

Occasional difficulty arises when the person wishes to travel urgently by air and has only just been vaccinated for the first time, so that his certificate is not yet valid. The airline might accept him as a passenger, if he completes a form of indemnity against liability for delays, expenses, etc. He would, however, have to take the risk of being refused admission or being detained in quarantine at his destination, though he might get in on compassionate grounds. Emergency travel would probably not be by sea and in any case would allow time for vaccination by the ship's surgeon.

The main facts about immunisation are given in Figure 70. Additional points are (1) The periods of validity may vary in countries not bound by the International Sanitary Regulations. (2) The minimum interval between doses of T.A.B.C., typhus and plague vaccines is seven days. (3) Parents consent for inoculation is necessary in the case of children under 18 years of age.

Sequence of Inoculations

Inoculations may have to be given as quickly as possible in order to allow certificates to become valid before departure. The following sequence practised by B.O.A.C. is convenient.

Day	Vaccine
1st	Anti-yellow fever (0.5 ml.). Anti-cholera (1 ml. of 8,000 million vibrios). Anti T.A.B.C. (1st dose—0.25 ml.).
4th	Vaccine-lymph against smallpox.

GUIDE TO IMMUNISATION FOR INTERNATIONAL TRAVEL

(Sir Harold Whittingham)

Immunisation against	Immunising Agent	Lower Age Limit	Dosage of Vaccine			Validity Period of Certificate	Booster Dose of Vaccine	Remarks
			Adults and Teenagers	Children				
(1)	(2)	(3)	(4)	1-5	6-12	(7)	(8)	(9)
SMALLPOX	Vaccine lymph	None	(a) 10 multiple pressures	(5)	(6)	(a) 8 days to 3 years from date of primary vaccination	Re-vaccination every 3 years by 30 multiple pressures	(a) Primary vaccination should be subsequent to yellow fever inoculation
	(b) Re-vaccination		(b) 30 multiple pressures			(b) For 3 years from date of vaccination		(b) Re-vaccination should normally be done 4 days after yellow fever inoculation, but, if done first, must precede yellow fever inoculation by 7 days
YELLOW FEVER	Attenuated strain of pantropic virus 17D vaccine	None	0.5 ml.	0.5 ml	0.5 ml.	10-12* days to 6 years from date of inoculation	0.5 ml. every 6 years	Should precede primary vaccination by at least 4 days. If given subsequent to primary vaccination there must be an interval of 21 days
	1,000 Mouse units per ml.							
CHOLERA	Vibrio cholerae vaccine 8,000 millions per ml.	1 year	1.0 ml.	0.2 ml	0.4 ml	6 days to 6 months from date of inoculation	1.0 ml. 6-monthly with appropriate body weight dose for children	

Immunisation against	Immunising Agent	Lower Age Limit	Dosage of Vaccine			Validity period of Certificate	Booster Dose of Vaccine	Remarks
			Adults and Teenagers	Children				
(1)	(2)	(3)	(4)	1-5 (5)	6-12 (6)	(7)	(8)	(9)
TYPHOID	(i) Alcoholised T.A.B.C. vaccine containing T. 1,000 millions per ml. A. 500 millions per ml. B. 500 millions per ml. C. 500 millions per ml. (ii) Non-alcoholised vaccine of similar number of organisms	1 year	Dose: (1st) 0.25 ml. (2nd) 0.5 ml.	0.1 ml. 0.2 ml.	0.2 ml. 0.4 ml.	Not an international requirement	0.25 ml. yearly	Is a recommendation only
PLAGUE	Pasteurella pestis vaccine 2000 millions per ml.	1 year	Dose: (1st) 0.5 ml. (2nd) 1.0 ml.	0.1 ml. 0.2 ml.	0.2 ml. 0.4 ml.	Not an international requirement	0.25 ml. yearly 1 ml. yearly with appropriate body weight dose for children	Only done where it is a specific requirement by the country of destination or transit. Is not a normal international requirement
TYPHUS	Epidemic and murine virus vaccine cultured in yolk-sac	1 year	Dose: (1st) 1.0 ml. (2nd) 1.0 ml.	0.2 ml. 0.2 ml.	0.4 ml. 0.4 ml.	Not an international requirement	1 ml. yearly with appropriate body weight dose for children	Only done where it is a specific requirement by the country of destination or transit. Is not a normal international requirement

* In Ceylon, India and Pakistan the validity period for yellow fever is 12 days to 6 years.

FIG. 70

7th - 9th Anti T.A.B.C. (2nd dose—0.5 ml.), also read results of primary vaccination against smallpox and enter on the International Certificate whether it is “ successful ” or not. The result of re-vaccination against smallpox has not got to be recorded.

Anti-yellow fever vaccination should always precede smallpox vaccinations by four days as, if smallpox vaccination was done first, 21 days would have to elapse before giving yellow fever vaccine. Smallpox and yellow fever inoculations should never be done at the same time. Yellow fever inoculation does not cause any reaction.

A passenger is liable to be vaccinated compulsorily or detained in quarantine if his certificates are not in order. Conscientious objectors are treated similarly. If for medical reasons the doctor decides that inoculations are inadvisable he should certify this, but his certificate is no guarantee against the passenger being quarantined.

LUMBAR PUNCTURE

(For hazards see page 21)

Position.

The patient should lie on his left side with his buttocks and shoulders on the hard edge of the bed (Fig. 71). If the mattress sags, put fracture boards under it. The long axis of the spine should be horizontal, and the plane of the iliac crests vertical. The spine must be fully flexed and the patient should be asked to get the chin as near to the knees as possible. A roller towel placed round the neck and knees, and tightened by twisting with a rod sometimes helps to obtain and maintain the flexed position.

Site of Puncture.

The usual site is the space between the spines of the 3rd and 4th lumbar vertebræ (*see page 21*). This space is on a plane passing through the highest points of both iliac crests.

The Puncture.

If difficulty is expected a stout (Barker) needle, or a nickel needle (*see page 16*) should be used. For routine use a smaller needle is better. Punctures must be made under full aseptic precautions. A finger-grip in front of the orifice, as in Dattner's

needle (Fig. 74, page 525) avoids the need of holding and perhaps contaminating, the shaft. Everything should be dry including the operator's hands. Gloves are not essential and instead of them a sterile towel may be used through which, the needle is held and the skin palpated.



FIG. 71

Position for lumbar puncture. The nurse places one arm under the knees and the other round the back of the neck in order to flex the spine as fully as possible.

A wheal of 2 per cent. procaine is raised in the skin over the junction of the lower and middle thirds of the interspace. The skin is pierced here by giving the lumbar puncture needle a rolling motion. The direction of the needle is then readjusted so that while in the horizontal plane it is inclined about 5 degrees towards the head. It is then parallel to the slope of the vertebral spines. No resistance is felt until the ligamentum flavum is reached. This is pierced with the bevel in the spinal axis (see page

22). The dura is next encountered and penetrated. Withdrawal of the stylet allows C.S.F. to flow.

Difficulties.

- (1) No fluid flows. Advance the needle a few millimetres and rotate it in case a nerve root is obstructing it.

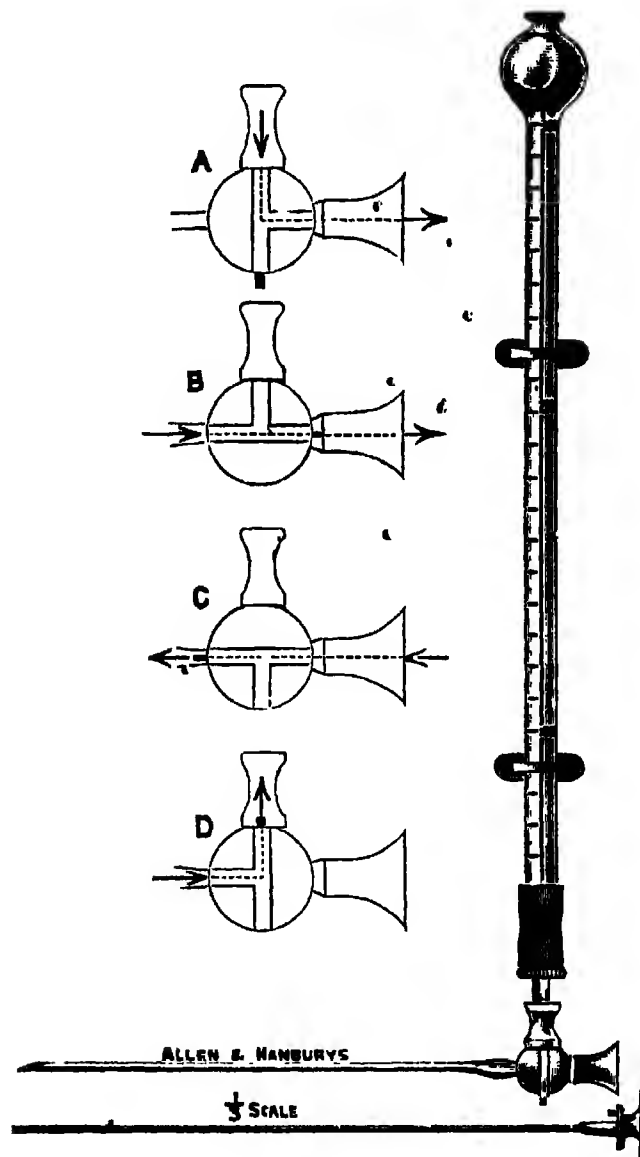


FIG. 72

Greenfield's spinal manometer.

- (2) Bone is encountered. This usually means that flexion is incomplete. Withdraw the needle and adjust the position.
- (3) Blood appears. If it is only a few drops and then nothing more, it means that the needle has not gone far enough but is in the subdural space. By over enthusiastic insertion, one may encounter the anterior subdural space.

If the fluid is bloodstained the decision must be made as to whether the "bloody tap" is due to trauma or subarachnoid hæmorrhage. Traumatic bleeding often clears as the fluid drains.

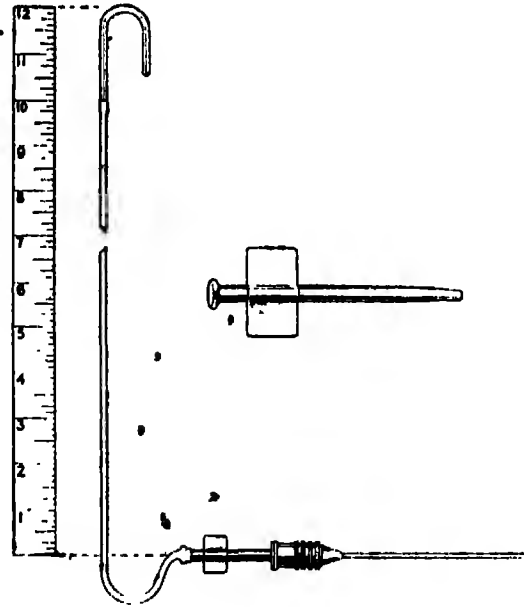


FIG. 73
Northfield's apparatus for measuring the cerebro-spinal fluid pressure.

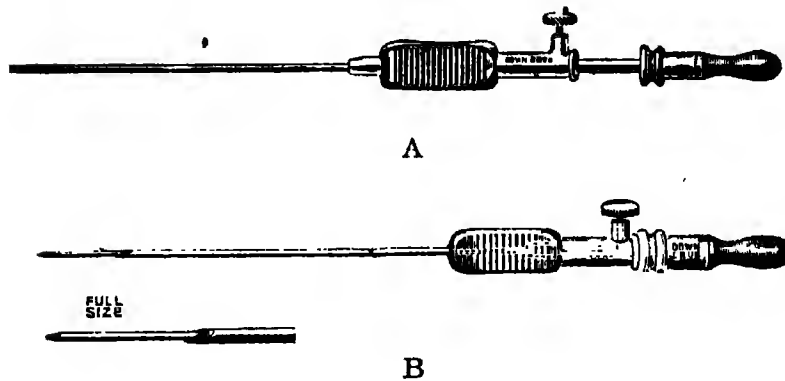


FIG. 74
Dattner's needle.

- A Arranged for introduction;
- B With needle projecting after introduction.

If in doubt, take specimens in three numbered tubes and have red cell counts done on all three. A diminishing count indicates traumatic blood. Clotting in the fluid may occur if bleeding was due to trauma, and if centrifugalised the supernatant fluid is colourless. In subarachnoid hæmorrhage clotting does not occur and the supernatant fluid is yellow.

Measuring the C.S.F. pressure.

This cannot be done by observing the rate of flow. A manometer, such as Greenfield's (Fig. 72) must be attached to the needle. A simpler method is to insert into the needle a butterfly adaptor with a length of bicycle valve tubing attached and at the other end of which is a small glass "U" tube. This is held below the level of the needle until fluid appears in it, and then raised until the fluid in one of its limbs is steady. Since CO_2 retention raises C.S.F. pressure it is advisable to wait until breathing is normal. A metal ruler with its zero mark level with the needle serves to read the pressure (Fig. 73).

Note the resting pressure and its response to coughing and straining. Failure to respond means that the needle is not in the correct place (or that there is complete block). Compress the right and left internal jugular veins separately and then together and record results (Queckenstedt's test).

The Dattner (double) needle.

This is useful if lumbar puncture has to be performed in cases of raised intracranial tension, and also to minimise the risk of post-puncture headache.

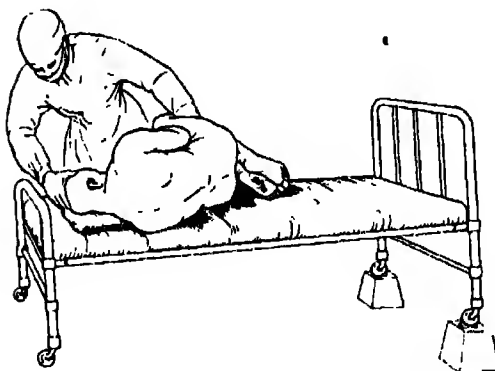


FIG. 75

Position for cisternal puncture in a comatose patient.

It (Fig. 74) consists of a fine needle and stylet inside a larger needle. The outer needle is inserted down to the ligamentum flavum, and the inner needle punctures the dura. If used carelessly the outer needle may penetrate the dura and the advantage of Dattner's principle will be lost. If correctly performed, fluid should cease to flow when the inner needle is with-

drawn into the outer case. A syringe must be used to withdraw C.S.F. Manometry is not possible.

CISTERNAL PUNCTURE

(For hazards see page 22)

Position.

The best position for a comatose patient is lying on the left side with his head at the foot of the bed so that the bedpost is

not in the way. The head is supported on a small sandbag and flexed. The spine should be horizontal (Fig. 75). The sitting position may be used in the conscious patient.

Site of puncture.

This is determined by the fact that a horizontal plane through the tips of the mastoid processes bisects the atlanto-occipital space

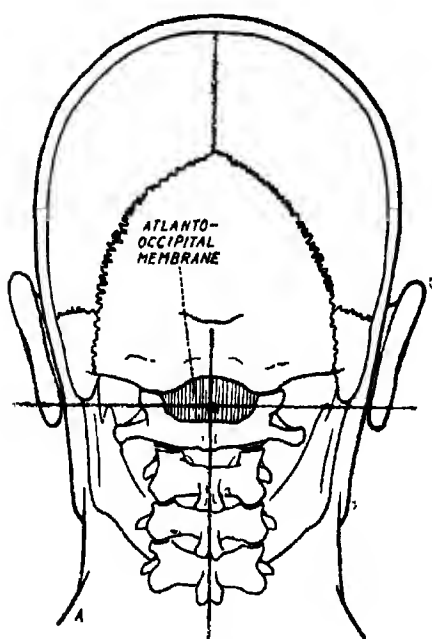


FIG. 76A

The point of entry of the needle at the intersection of a line joining the tips of the mastoids and the vertical midline of the neck.

(*"Pye's Surgical Handicraft."*)

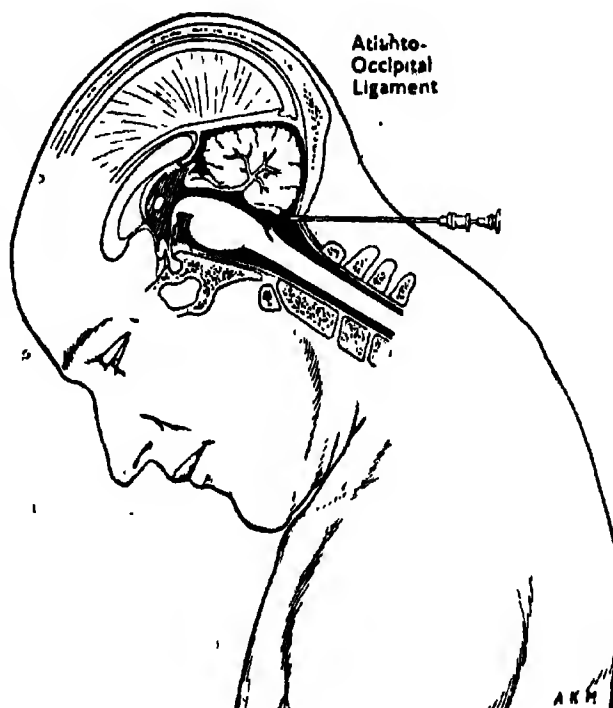


FIG. 76B

Showing the direction of the needle which has reached the atlanto-occipital ligament.

(*Surgery of Modern Warfare*)

(Fig. 76A). The tips of the mastoid processes are marked, and a horizontal line joining them is drawn using a tape measure and a grease pencil. The point where this line crosses the vertical midline of the neck is marked. This is the entry point.

The puncture.

A Purves-Stewart graduated needle (Fig. 77) is convenient but a lumbar puncture needle may be used. A scratch made on it 5 cm. from the tip is a useful guide.

The needle is directed slightly upwards so that the occipital bone is hit (Fig. 76B). It is then worked down tapping the bone periodically until the atlanto-occipital ligament is reached. When this is pierced there is a characteristic "give." The needle is then

advanced cautiously, removing the stylét at intervals until fluid appears. Some operators prefer to mount the needle on a syringe and apply slight suction when the atlanto-occipital ligament is pierced.

A rough idea of the depth at which the ligament will be reached is given by the neck circumference at the level of the

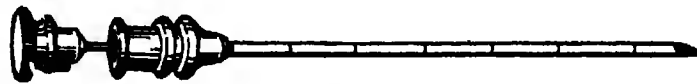


FIG. 77

Purves-Stewart's cisternal puncture needle.
(*"Pye's Surgical Handicraft."*)

upper border of the thyroid cartilage divided by nine. In an adult the cistern is about 2 inches from the skin and the medulla is about half an inch further on.

STELLATE GANGLION BLOCK

The object is to infiltrate with local anæsthetic the region of the stellate ganglion on the side of the affected cerebral hemisphere (*i.e.*, the opposite side to the paralysed arm and leg). The anterior route is the safest since the needle is above the dome of the pleura and there is little danger of pneumothorax. The injection is aimed at the upper part of the ganglion and we rely on procaine seeping down to the part of the ganglion which overlies the neck of the first rib. The entry point for the needle is a centimetre below the level of the lower border of the body of the 6th cervical vertebra close to the lateral border of the trachea. Two landmarks indicate the 6th cervical vertebra. The cricoid cartilage lies opposite the body and the large anterior tubercle of its transverse process (Chassaignac's tubercle) is fairly easy to feel. The entry point can also be marked from below being about 4 cm. above the inner end of the clavicle at the median border of the sterno-mastoid muscle. Raise a wheal of 1 per cent. procaine at this point. Press the left middle finger deeply on the inner border of the sterno-mastoid muscle and palpate and displace laterally the carotid artery. Insert a 4-inch needle along the finger and through the wheal straight backwards between the carotid artery and the trachea until bone (the body of the seventh cervical vertebra) is felt. These relationships are shown in Figure 78. The stellate ganglion does not lie on the periosteum but on the

longus colli muscle and so the needle should be withdrawn 0.5 to 1.0 cm. and a negative aspiration test obtained before injecting 5 ml. of 1 per cent. procaine. After withdrawing the needle a further 1 cm. 5 ml. more are injected. The patient should be told to resist the desire to cough. Pain may be felt in the chest and arm but will pass off when the procaine is injected.

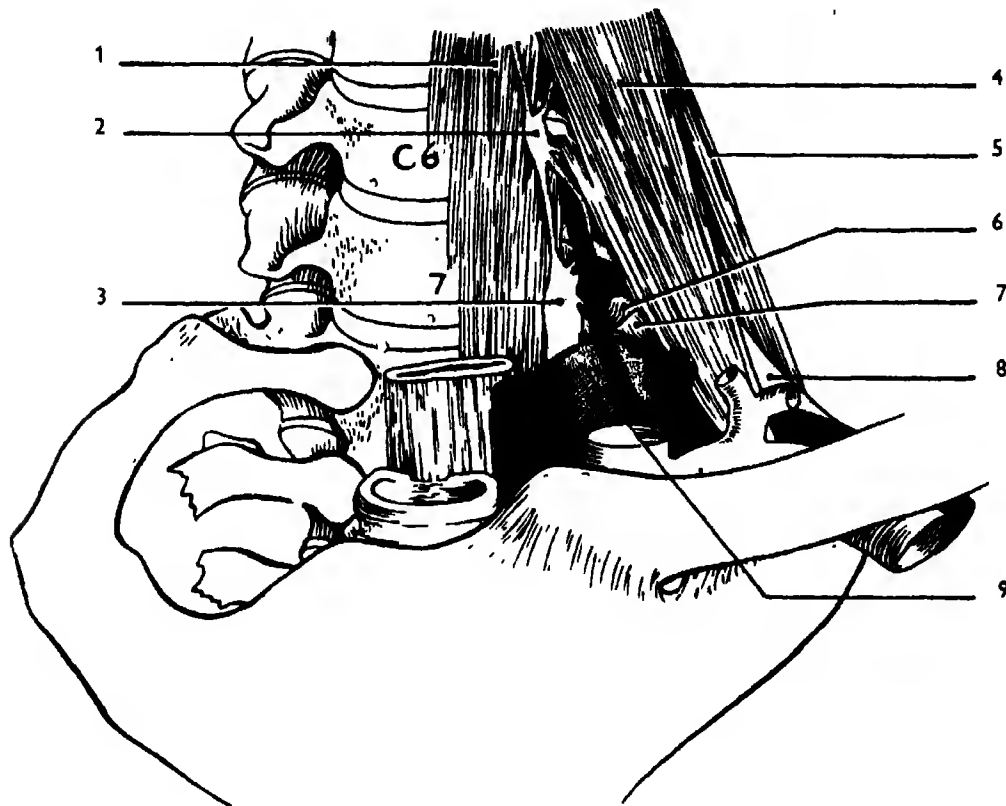


FIG. 78

The relations of the stellate ganglion.

1. Longus colli muscle. 2. Middle cervical ganglion. 3. Stellate ganglion. 4. Scalenus anterior muscle. 5. Scalenus medius muscle. 6. Transverse process of first thoracic vertebra. 7. Tubercle of first rib. 8. Brachial plexus. 9. Dome of Pleura.

A successful injection is followed in about 10 minutes by Horner's syndrome (ptosis, miosis, enophthalmos, flushing of the face and conjunctiva and possibly unilateral blocking of the nose from engorgement). When performed for cerebral thrombosis or embolism the injection should be repeated every six hours during the first day and then daily for up to 15 days according to progress. It is possible to thread a fine polythene tube down the needle and to leave it in place for subsequent injections.

PARACENTESIS OF THE PERICARDIUM

There are three possible routes—anterior, epigastric and posterior.

- (1) **Anterior.** The needle is inserted (a) in the fifth left interspace just outside the apex beat but inside the outer edge of the cardiac dulness, or (b) in the fourth left interspace one inch from the sternal margin (to avoid the internal mammary artery). The right side should be avoided lest the right auricle be injured.

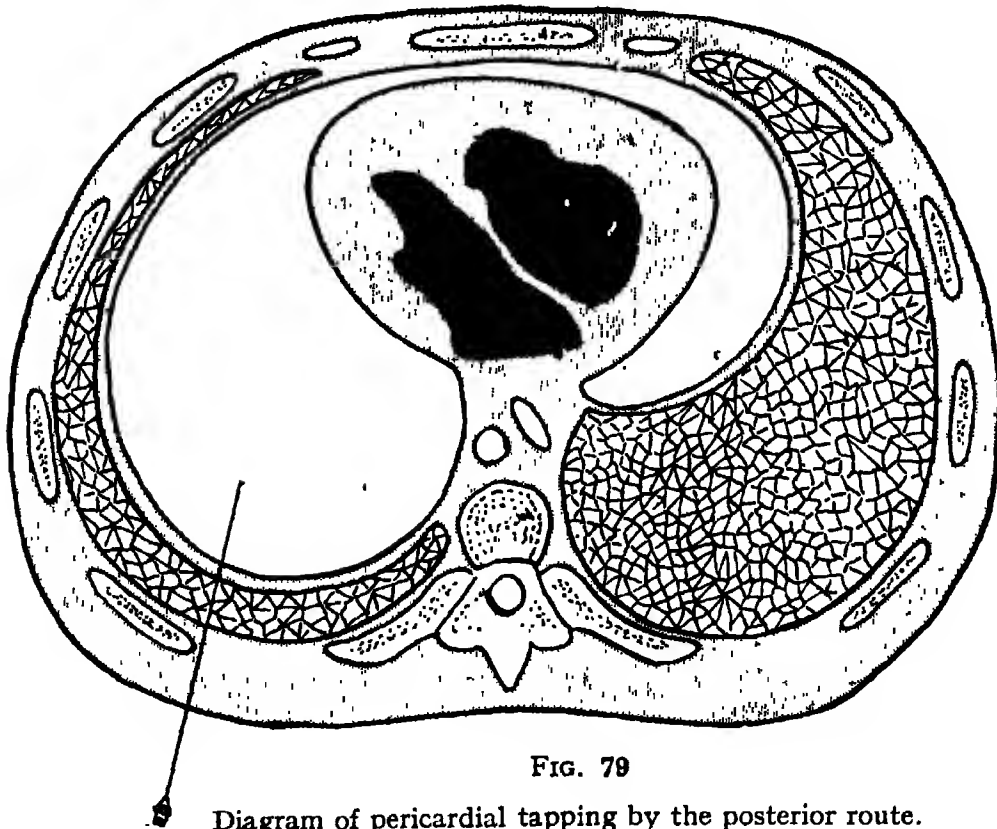


FIG. 79

Diagram of pericardial tapping by the posterior route.

- (2) **Epigastric.** The needle is inserted at the costo-xiphoid notch and directed upwards and backwards to enter the lowest part of the pericardial sac.
- (3) **Posterior.** The needle is inserted near the inferior angle of the scapula on the left side. It traverses the lung to reach the pericardium (Fig. 79).

The skin is cleansed, and the skin and subcutaneous tissues infiltrated with 2 per cent. procaine. A long needle of about 1 mm. bore is used for the pericardial tap. The anterior routes are safer than the posterior, but it is not possible to remove large

quantities of fluid by them. When an effusion is very large it collects posteriorly and compresses the lung (Fig. 79). In such a case the posterior route is best. When empyema of the pericardium is suspected, approach through the pleura and lung is contraindicated and the epigastric route, using a fine trocar and cannula, is to be preferred. It is probably wise in such a case to enlist the help of a surgeon.

ARTIFICIAL PNEUMOTHORAX INDUCTION

In young or sensitive patients sedation is advisable and Linctus of Codeine B.P.C. should be given if there is a cough.

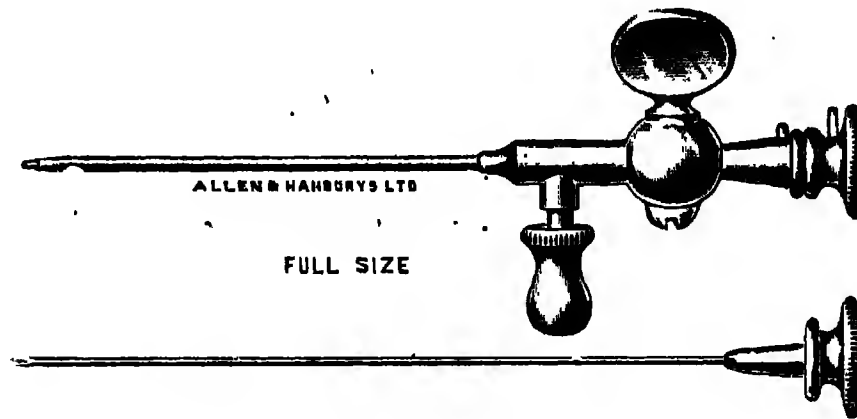


FIG. 80
Kuss Needle.

The patient lies on the "good side" with his upper arm raised to expose the axilla. With aseptic precautions the skin and tissues down to the pleura are infiltrated with 2 per cent. procaine in the fourth, fifth or sixth inter-costal spaces in the mid-axillary line.

A needle with a flat end and sharp trocar such as the Kuss needle (Fig. 80) attached to an artificial pneumothorax apparatus and open to the manometer is inserted down to the pleura. It is best to choose a spot at the upper border of a rib so as to avoid the vessels in the sub-costal groove of the rib above. The sharp trocar is then removed. Very often a sharp "hiss" as air rushes in shows that the pleura has been entered. If it has not, the blunt trocar should replace the sharp one before further insertion. A good negative swing of the manometer fluid in both phases of respiration indicates that the needle is in the pleural space. The

clips are then turned on and air is drawn into the chest. The usual amount for induction is 100 to 300 ml.

Failure to enter the pleural space is shown by the behaviour of the manometer:—

- (1) A small equal swing on each side of zero means that the needle is in the lung tissue. A similar but larger swing indicates that it is in a lung cavity.
- (2) A rising positive pressure means that the needle has punctured a blood vessel. It should be withdrawn promptly.
- (3) Absence of any reading means that the point of the needle is in the chest wall, fluid, or an adhesion, or that the tubing or needle is blocked. But a small negative swing of the order of -1 -3 is sometimes obtained when the needle point is situated extra-pleurally.

In the case of failure, other sites should be tried before abandoning the procedure, such as the 7th or 8th spaces posteriorly, and the 2nd, 3rd, and 4th spaces anteriorly.

Numerous types of apparatus exist, the earlier ones consisting simply of two bottles and a manometer. When using an apparatus of this type for an induction, the fluid levels in each bottle should be at the same height. The negative intra-pleural pressure draws air into the chest. If the fluid levels are different, air enters the chest under pressure and the risk of air embolism is increased. In some machines the manometer is open to the needle all the time. In others a tap or clip has to be opened but with all, the pressure changes must be carefully observed.

PNEUMO-PERITONEUM INDUCTION

The most convenient site for the intra-peritoneal injection of air is near the left subcostal margin just external to the rectus abdominis muscle.

With aseptic precautions a wheal of 2 per cent. procaine is raised in the skin with a small hypodermic needle. The syringe (2 ml.) is recharged and, using a long serum needle, the abdominal wall is infiltrated down to the peritoneum. When this is punctured the sense of resistance to the piston disappears. Sudden penetration should be avoided. The piston should be withdrawn to make sure that the needle point is not in a blood vessel. A pneumothorax apparatus is then attached to the needle by an adaptor. Air is slowly injected in amounts up to 1,000 ml.

A manometer is unnecessary, but if available provides a useful indication that the needle is in the correct space by showing respiratory excursions and also a rise when the abdomen is pressed on. If before the air is run in the clips are suddenly opened and closed, the fluid in the manometer will rise and fall rapidly to almost zero if the peritoneal space has been entered. If the needle is outside the peritoneum, the pressure will remain high.

Some operators prefer a pneumothorax needle,* and others a special Veress needle. This consists of a sharp needle in which runs a hollow, blunt-ended trocar with a lateral hole. The trocar is made to project slightly from the needle by a spring, thus ensuring that the gut is pushed away by the blunt end rather than the sharp needle point.

Clinical evidence that air has entered the peritoneal space is provided by:—

- (1) Pain in the shoulder regions (but not on the side on which the phrenic nerve has been interrupted).
- (2) Disappearance of liver dullness.

The most frequent cause of failure to induce a pneumoperitoneum is that the needle is not inserted far enough.

CONTINUOUS SUCTION OF PNEUMOTHORAX

(For indications see page 137)

Several methods are available.

(1) **Sprengel's pump using water.**

The patient's bed is moved near to a water tap to which a Sprengel's filter pump is attached. This is connected to a needle in the chest (Fig. 16, page 138). A water bottle should intervene to indicate that air is being aspirated and a manometer should be connected to a side arm of the needle.

(2) **Sprengel's pump using oxygen.**

Using a filter pump with a very fine jet, a similar apparatus can be set up using oxygen instead of water (Fig. 81). It is wise to protect the outlet of the pump by wire gauze.* A flow of 4 litres per minute will produce a negative pressure of 8 ml. of water.

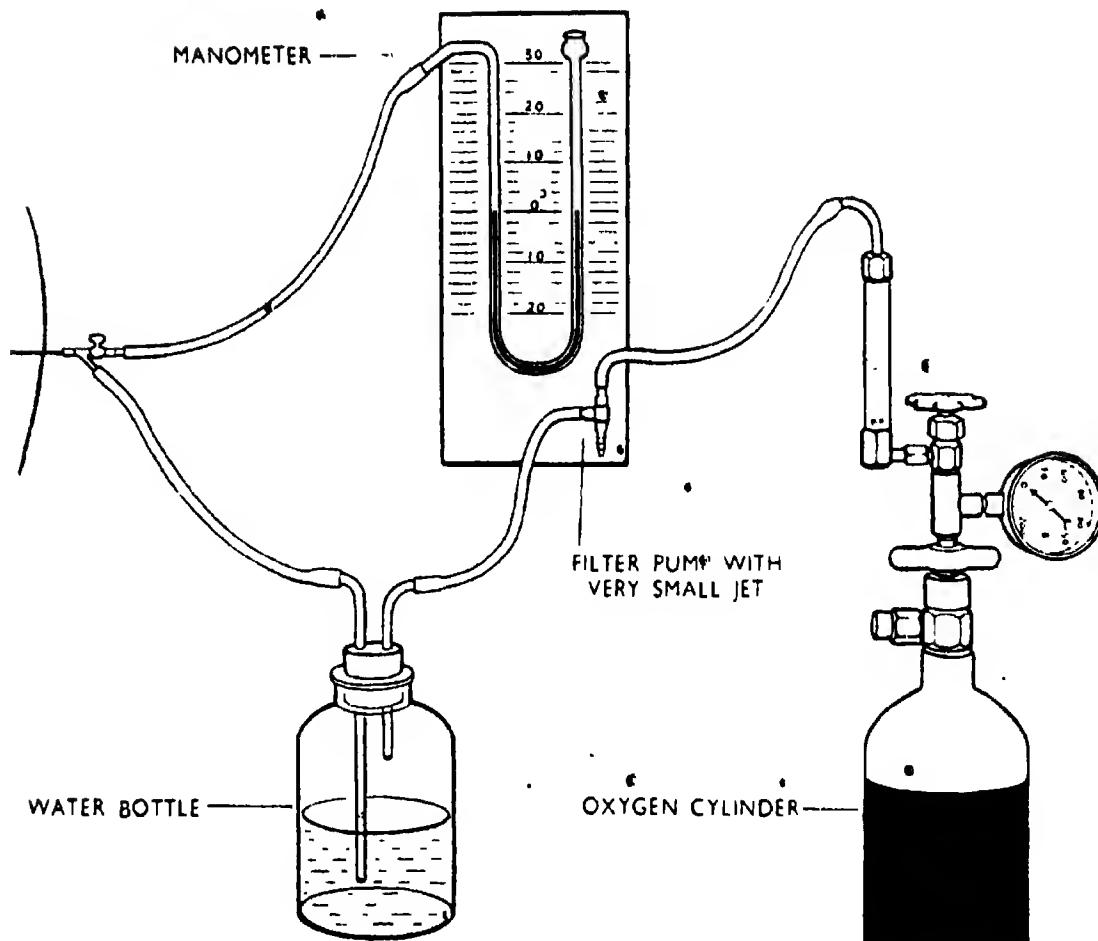


FIG 81

Diagram of apparatus for continuous suction of pneumothorax.

(3) Marriott and Foster-Carter's apparatus.

This apparatus consists of a large bottle, A (Fig. 82), capable of holding at least 80 fl. oz. (2.25 litres approx.) and having at the bottom an outlet, B, provided with a tap. This outlet tube must be straight and not curved downwards, otherwise it may act as a siphon. The neck of the bottle is closed with a rubber cork, pierced by a tube E, which dips below the level of the water in A and can slide in the cork, so that its height may be readily altered. When tap B is opened, water will flow from bottle A, and air will be drawn in through tube E. If E is closed, or if it communicates with a closed cavity, a negative pressure will develop within it, proportional to the height of its lower end X above the level Y of the outflow. Thus, if the distance XY is 10 cm., a negative pressure of 10 cm. of water will develop in E when tap B is opened.

When used in the treatment of spontaneous pneumothorax, the suction tube E is connected by rubber tubing with a pneumothorax needle. It is also convenient to incorporate a water manometer in the system to measure both the intra-pleural pressure when clip C is closed and the negative pressure developed by the apparatus when clip D is closed. Bottle A should be refilled with clip C closed, before the water falls below the level of X. A slight disadvantage of this apparatus is that, if the leak is large, the bottle has to be filled frequently.

(4) An electrical suction pump.

This should be capable of keeping up a low continuous suction.

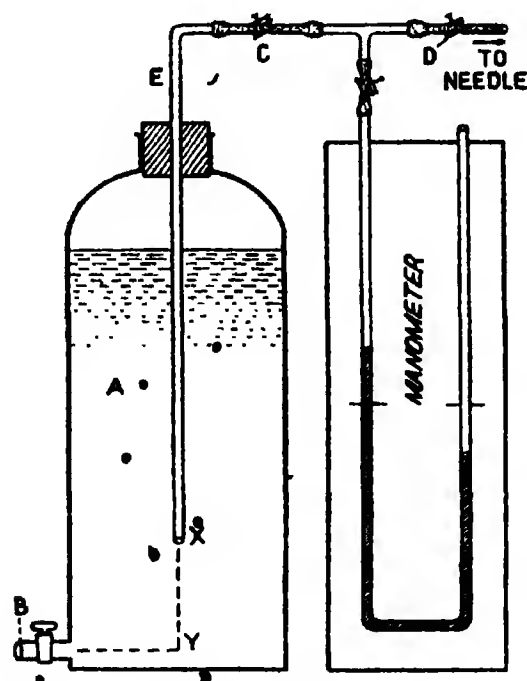


FIG. 82

Apparatus for continuous suction of pneumothorax.

TIDAL IRRIGATION OF THE PARALYSED BLADDER

The principle is that the bladder is slowly filled with a mild antiseptic solution from a container and automatically emptied at intervals by siphonage.

The irrigating fluid drips into the outer chamber (Figs. 83 and 84) and also passes to the bladder and into the ascending limb of the U-tube. When the fluid level in the outer chamber reaches the bend of the U-tube, the latter forms a siphon sucking out the contents of the bladder and of the outer chamber, which pass to the container. The height of the bend of the U-tube above the symphysis pubis determines the final pressure reached in the bladder. This should not be more than a few inches for completely paralysed bladders. As bladder tone improves, less fluid will be needed before siphonage begins and, when the bladder is able to empty itself reflexly, tidal irrigation can be discontinued. If, however, the residual urine is large in amount after reflex emptying, tidal irrigation should be restarted until reflex evacuation is more complete.

Note :

1. The rubber tube to the container should not lie under a considerable depth of fluid, as the increased pressure will be projected back to the patient's bladder.

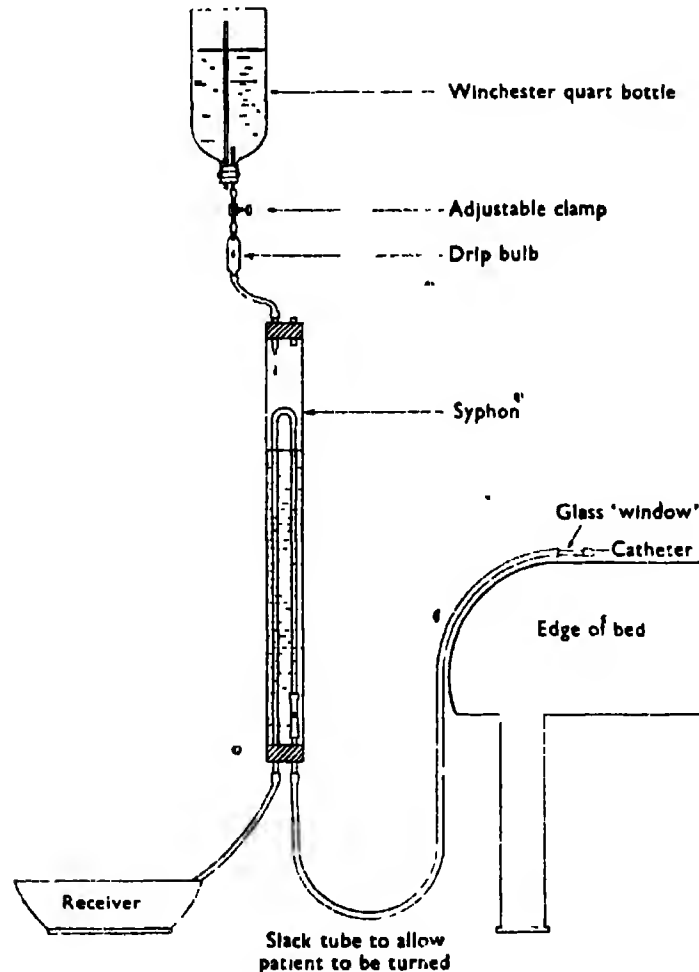


FIG 83

Apparatus for tidal irrigation of the bladder

2. Not less than 2 gallons (16 pints or 8 litres approximately) of irrigating fluid should pass through the apparatus in 24 hours, i.e., a Winchester "quart" bottle (4 pints or 2 litres approximately) should be used every six hours and an ordinary drip bulb will have to be *kept* running at two drops per second to deliver this amount.
3. In severe cystitis clots of pus may block the U-tube. In this case the outer tube will overflow through the air hole.

Supra-pubic route.

To avoid the disadvantages of the indwelling urethral catheter in the above method on the one hand and a supra-pubic cysto-

tomy on the other, tidal drainage may be applied through a small leakproof supra-pubic catheter. This requires a special technique for its introduction (Riches, E. W., *Lancet*, 1943, 1, 128).

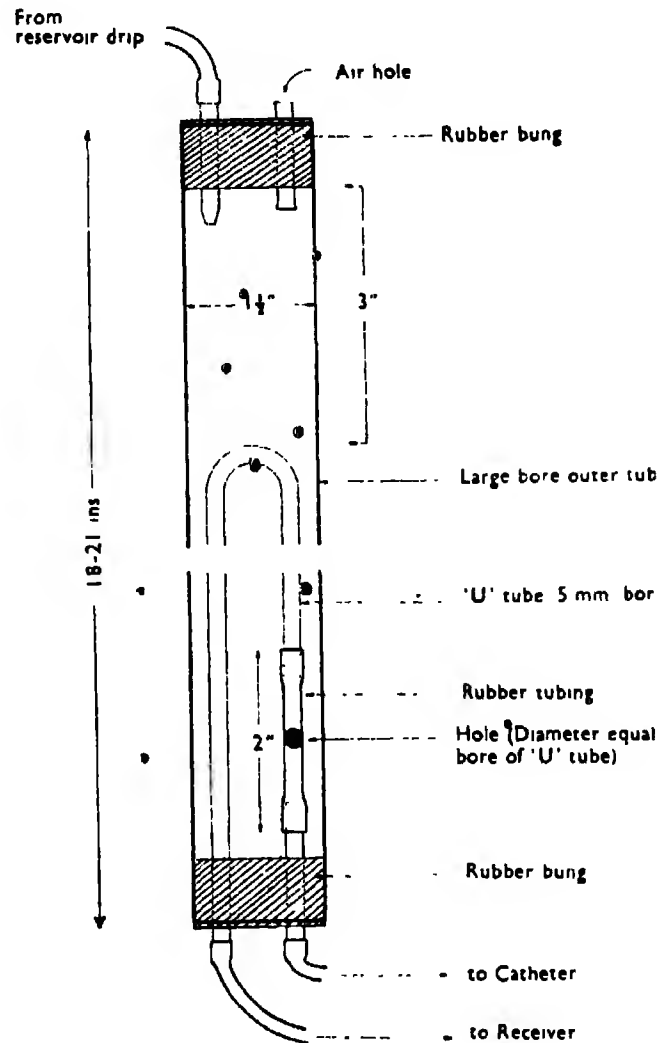


FIG. 84

Showing details of the syphon chamber.

GASTRIC LAVAGE

Position of patient.

In an unconscious patient with impending respiratory failure the insertion of a cuffed endo-tracheal tube by an anæsthetist is a useful preliminary to gastric lavage. Failing this the risk of fluid entering the lungs should be avoided by having the head low. A cuffed œsophageal tube is an additional safeguard. The patient

should be placed prone with his head over the table (Fig. 85), or in the Trendelenburg position on an operating table (Fig. 86). In the latter position, it is necessary to draw the tongue forwards



FIG. 85

Gastric lavage on an operating table. A. Stomach tube
B. Large safety pin in wall of tube. C. Gag. D. Sucker
tube. E. Tongue forceps F. Electric sucker

by a clip and to remove fluid from the mouth by a sucker. A struggling patient may have to be immobilised by straps, or by wrapping him tightly in a blanket.

Technique.

Dentures are removed and the mouth opened by a gag or box-wood wedge with a central hole (Fig. 87). A fairly stiff oesophageal



FIG 86

Gastric lavage, showing the proper position of the patient. The head must be lower than the rest of the patient. A: Stomach tube. B. Safety pin in wall of tube. C. Gag.

tube (not a Ryle's tube), preferably 60 inches long and about half an inch in diameter (for an adult) is lubricated, several large holes having been cut in its wall near the end. It is then passed

through the hole in the wedge if this is used, over the tongue and quickly down the œsophagus. The end of the tube should be 20 inches from the incisor teeth in an adult, and 10 inches in an



FIG. 87

Boxwood wedge with central hole to take œsophageal tube.



FIG. 88

" Puretha " respirator.
(Siebe, Gorman and Co. Ltd.)

infant. It is an advantage to mark these distances on the tube by a safety pin in its wall (but not in its lumen). A Ryle's tube should only be used if it is impossible to open the mouth. It may then be passed through the nose.

When the tube enters the stomach it is useful first to attach a Senoran's evacuator (Fig. 89) to empty the stomach. Then a funnel is attached and warm water is poured in. Bicarbonate solution may be used but not in barbiturate poisoning (*see page*

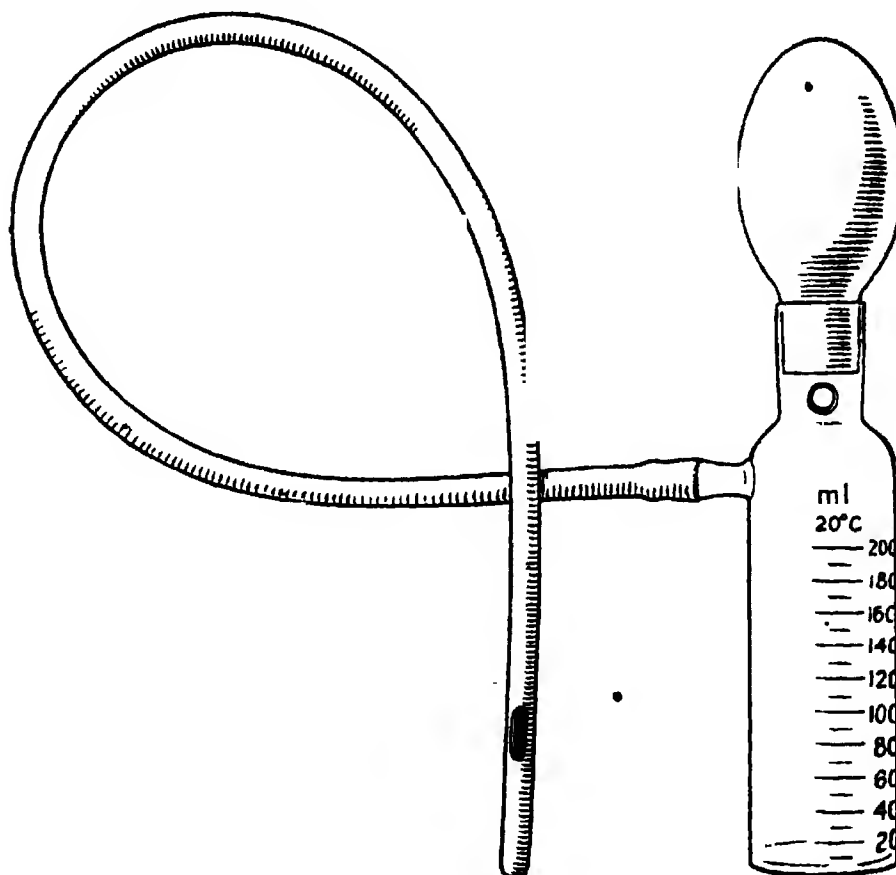


FIG 89
Senoran's evacuator.

11). After about half a pint has entered (less in children) but while the level of water is still visible in it, the funnel is lowered over a pail on the floor, and the fluid siphoned out of the stomach. Larger amounts may force the pylorus and so defeat the object of gastric lavage. A total amount of two gallons should be used, and the first washings should be preserved separately in case analysis is required.

RESPIRATORS

Three main types are available for rescue purposes. At least two models appropriate to the industry concerned should always be provided, and workmen should be practised in their use.

- (1) Canister types such as the "Puretha" (Fig. 88). These can be relied on for short periods in low concentrations of gas. Numerous canisters of distinctive colour are provided to give protection against different gases. Appropriate canisters should be available in any given industry.



FIG. 90

"Antipoy's" short distance breathing apparatus.
(Siebe, Gorman and Co. Ltd.)

- (2) Fresh air apparatus. "The Antipoy's" (Fig. 90). This has the same facepiece as the "Puretha" respirator and is connected to an air pipe 30 feet (9 metres approx.) long, the end of which must be in fresh air.
- (3) Self-contained oxygen apparatus. "The Salvus" (Fig. 91). This apparatus enables the wearer to remain up to half an hour in an atmosphere of 100 per cent. irrespirable air. It delivers oxygen automatically at 2 litres per minute and there

is a relief valve to deflate the breathing bag should this become over-inflated. Eye protecting goggles should be worn also in smoke or gases affecting the eyes.



FIG. 91

"Salvus" self contained oxygen
breathing apparatus.

(Siebe, Gorman and Co. Ltd.)

ARTIFICIAL RESPIRATION

Sudden failure of respiration demands prompt treatment, for if breathing has stopped for ten minutes death is almost certain and may even occur after a two-minute stoppage. After rescue from drowning or electrocution artificial respiration must be started at once. *Delay is dangerous and there is literally not one second to lose.* Don't stop to remove dentures, loosen clothing or drain the lungs. All these can be attended to later or by an assistant.

No one method of artificial respiration is universally applicable and the choice will depend on which one the operator knows, whether he is single-handed or not, and how much space is available. "Push and pull" methods are twice as effective as simple "push" methods, since they cause active inspiration as well as expiration. Success depends on the time factor, and so the best method is whichever the operator can promptly and persistently apply. Don't hurry with artificial respiration. If you do you will not only exhaust yourself but you may delay the onset of normal respiration and return to consciousness by lowering the CO_2 tension of the patient's blood through hyperventilation.

The main methods are:—

- (1) Holger Nielsen's arm lift - back pressure method (a "push and pull" method for a single operator). This causes better ventilation than other methods and also avoids the danger of pressure on the stomach causing regurgitation and inhalation of water. It is now recommended by the British Red Cross Society.
- (2) Schäfer's prone-pressure method (a "push" method for a single operator). This might have to be used if injuries to the arms and ribs prevented the use of the Holger Nielsen method. It is only for this reason that it is described here.
- (3) Drinker's arm-lift—a prone-pressure method. (Eve's modification—a "push and pull" method for two operators).
- (4) Mouth-to-mouth insufflation.
- (5) Eve's rocking method.

Holger Nielsen's method.

The chest is compressed against the ground for expiration and raised by the arms for inspiration.

1. Turn the patient face downwards with arms upwards and elbows flexed so that his head is turned to the side and rests on his hands.
2. Kneel on one knee at the patient's head and facing his feet. The opposite foot is near the patient's elbow (Fig. 92).
3. Inspiration. Grasp the arms above the elbows and rock backwards (Fig. 93) raising the arms until tension is felt. Count 1, 2, 3 while doing this ($= 2\frac{1}{2}$ seconds).

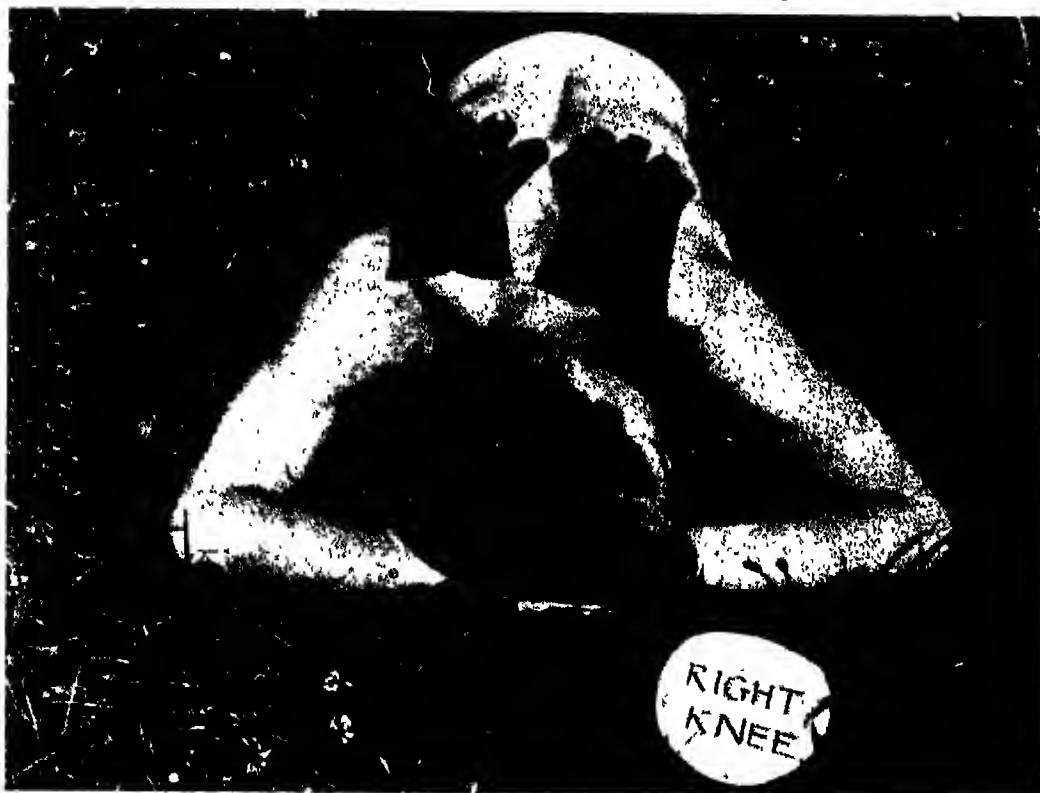


FIG. 92

Holger Nielsen method. Showing the position of the operator's hands (shown as gloves); his left foot (shown as a shoe); his right knee and also the correct position of the patient's arms and head.

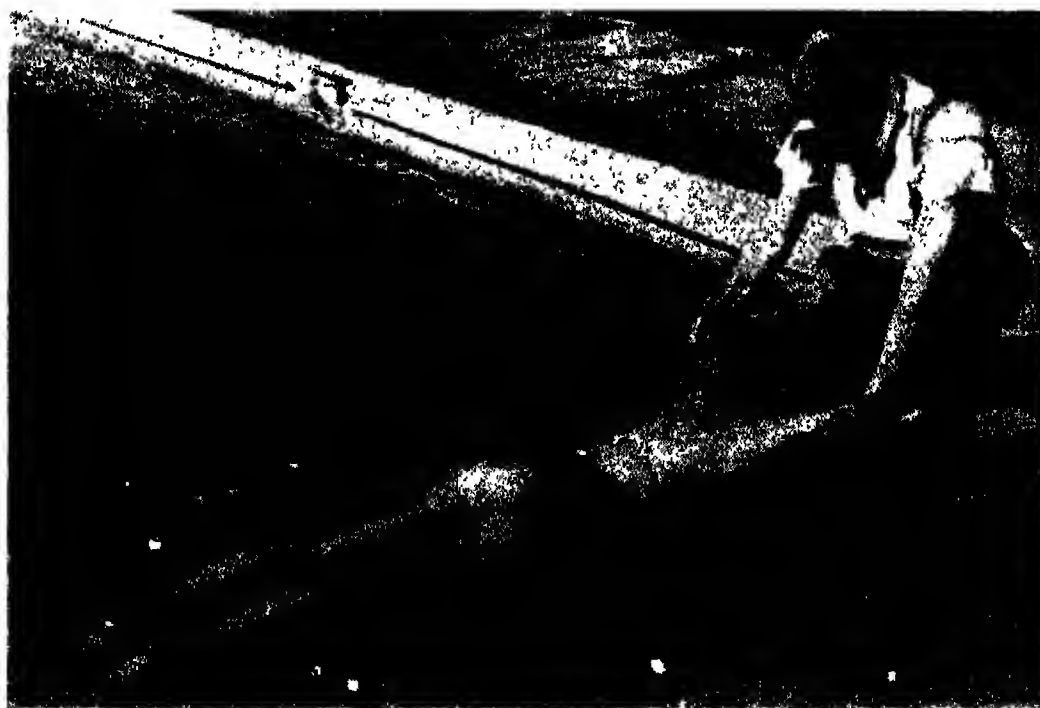


FIG. 93

Holger Nielsen method. Inspiration. Duration $2\frac{1}{4}$ secs.

4. Expiration. Drop the arms (Fig. 94) and put your hands on the patient's back just below the scapulæ with your thumbs touching (Fig. 95) counting 4, as you do it. Rock forwards



FIG. 94

Holger Nielsen method Patient's arms dropped at end of inspiration.



FIG. 95

Holger Nielsen method. Position of operator's hands and arms at beginning of expiration.

with your elbows straight and exert steady pressure on the chest (Fig. 96). Count 5, 6, 7 while doing this ($= 2\frac{1}{2}$ seconds).

5. Slide your hands off the back and on to the patient's arms as you count 8.

This cycle should be repeated 10 to 12 times a minute.

If the arm is injured inspiration should be achieved by lifting from the shoulders rather than from the upper arms.



FIG. 96

Holger Nielsen method. Expiration. Operator rocks forward with his arms straight. Duration $2\frac{1}{2}$ secs.

Schäfer's method.

1. Turn the patient face downwards with one arm extended overhead and the other bent at the elbow. His face is turned outwards and rests on his hand.
2. Kneel, straddling the patient's thighs or one thigh if this is too difficult, and place the palms of your hands on the small of his back with the tips of your fingers just out of sight.
3. Expiration. Keeping your arms straight swing forwards slowly so that the weight of your body is gradually brought to bear on the patient. This takes two seconds. (Count "one thousand, two thousand" or "one chimpanzee" and so on). Your shoulder should be directly over your wrist at the end of the forward swing.
4. Inspiration. Swing backwards for three seconds to remove the pressure completely. After one or two seconds swing forwards again.

The important point in Schäfer's method is that expiration depends on compression of the abdominal viscera pushing the diaphragm upwards. The operator should feel that the loins are yielding. His hands should therefore be placed low down over the small of the back and not over the rigid part of the thorax. They should not be taken off the patient during the whole time of the procedure. Inspiration is effected by the elastic recoil of the thorax and diaphragm. The disadvantages of Schäfer's method are that the operator cannot see what is happening so that it may degenerate into a ritual and also if the patient is deeply unconscious and cold the diaphragm will be toneless and unable to cause inspiration by elastic recoil. Inspiration should therefore be assisted by a second operator who lifts the arms (Drinker's method. See below). If there is a third assistant he should determine by feeling and listening that air is going in and out, and should also help by rubbing the limbs towards the heart. Relays of operators are necessary as the method is tiring.



FIG. 97

Drinker's arm-lift—prone-pressure method. Eve's modification. Expiration is produced by one operator using Schäfer's method



FIG. 98

Drinker's arm-lift—prone-pressure method. Eve's modification. Inspiration.

One operator using Schäfer's method takes pressure off the loins while the second operator assists inspiration by pulling up the patient's arms until the upper abdominal wall is off the ground. The patient's head is supported on the operator's knees,

Drinker's arm-lift—prone pressure method. (Eve's modification).

Professor Drinker suggested that inspiration (the weak point of Schäfer's method) could be improved by having a second operator to pull the arms towards the head as in the Danish method of Nielsen. Eve still further improved the method by having the arms *lifted* upwards (*i.e.* off the ground). The technique is as follows:—

One operator applies Schäfer's method for expiration (Fig. 97). The other kneels at the patient's head and assists inspiration by lifting the arms upwards (Fig. 98) so that the abdominal wall is just clear of the ground. This allows the upper abdominal contents to sag and causes the diaphragm to be pulled down.

Mouth-to-mouth method.

This ancient method mentioned in the Bible (II Kings IV: 34) consists in blowing up the lungs and while it provides adequate tidal air it is generally avoided as being æsthetically disagreeable. It is, however, very useful in children for asphyxia other than from drowning and in the newly born.

Compress the patient's nostrils with your right hand. Support his jaw with the fourth and fifth fingers of your left hand and with your thumb and other two fingers of the left hand cup his mouth. Considerable pressure between your lips and his is required. An airway is an advantage as the tongue may fall back.

Eve's rocking method.

Eve has pointed out that manual methods of artificial respiration depend on the elastic recoil of the thoracic wall and diaphragm for inspiration, and that this may be absent in the drowned because of loss of muscular tone. In such cases a rocking method is advisable. Besides causing efficient ventilation, it has the advantage of aiding circulation. Wet clothes can be replaced and dressings applied while rocking goes on. Relays of trained operators are not needed.

Rocking may be achieved in various ways:—

1. On specially designed stretchers or beds (Fig. 99).
2. On improvised rocking stretchers.

A door or ladder seven feet long is obtained and the patient lashed to it. For a trestle a low fence, or the backs of two chairs, or a loop of rope may be used—all of which should

CHAPTER XXVIII

Practical Procedures

WEIGHTS AND MEASURES

TO conform with modern measurements of doses these are all given in the "Metric" system but in the case of older drugs the approximate Imperial equivalent is also shown. In prescribing, however, it will be found convenient to use the *nearest familiar dose* in the "Metric" system to the dose in the Imperial system rather than the *exact equivalent*. For example, while in the United Kingdom we prescribe morphine gr. $\frac{1}{6}$ or $\frac{1}{4}$ (approximate metric equivalents 11 and 16 mg.) it is more usual in countries using the "Metric" system to prescribe 10 or 20 mg. When giving medicines prescribed in the Imperial system it is best to avoid domestic measures and to use a measuring glass because, although the British Standard specification for a medicinal teaspoon is 60 m or 3.6 ml., the size of the domestic teaspoon varies from 2.4 to 7.0 ml.

The following tables give the approximate "Metric" equivalents of Imperial doses. (See also Preface to Second Edition page vi).

WEIGHTS

Imperial	"Metric"	Imperial	"Metric"
1 oz.	30 G. (grammes)	$\frac{3}{8}$ gr.	24 mg.
75 gr. (grains)	5 G.	$\frac{1}{3}$ gr.	22 mg.
60 gr.	4 G.	$\frac{1}{4}$ gr.	16 mg.
45 gr.	3 G.	$\frac{1}{5}$ gr.	13 mg.
30 gr.	2 G.	$\frac{1}{6}$ gr.	11 mg.
15 gr.	1 G.	$\frac{1}{8}$ gr.	8 mg.
10 gr.	0.65 G.	$\frac{1}{10}$ gr.	6.5 mg.
$7\frac{1}{2}$ gr.	0.5 G.	$\frac{1}{12}$ gr.	5.4 mg.
7 gr.	0.45 G.	$\frac{1}{16}$ gr.	4 mg.
6 gr.	0.4 G.	$\frac{1}{20}$ gr.	3.2 mg.
5 gr.	0.32 G.	$\frac{1}{32}$ gr.	2 mg.
4 gr.	0.25 G.	$\frac{1}{64}$ gr.	1 mg.
3 gr.	0.2 G.	$\frac{1}{100}$ gr.	0.65 mg.
$2\frac{1}{2}$ gr.	0.16 G.	$\frac{1}{120}$ gr.	0.54 mg.
2 gr.	0.13 G.	$\frac{1}{160}$ gr.	0.4 mg.
$1\frac{1}{2}$ gr.	0.1 G.	$\frac{1}{200}$ gr.	0.32 mg.
1 gr.	65 mg. (milligrams)	$\frac{1}{250}$ gr.	0.26 mg.
$\frac{3}{4}$ gr.	50 mg.	$\frac{1}{320}$ gr.	0.2 mg.
$\frac{2}{3}$ gr.	45 mg.	$\frac{1}{640}$ gr.	0.1 mg.
$\frac{1}{2}$ gr.	32 mg.		

LIQUID MEASURES

Imperial	" Metric "	
1 pint (20 fl. oz.)	568	ml. (Millilitres) (Approx. 600 ml.)
12 fluid ounces	340	ml.
8 fluid ounces	227	ml.
6 fluid ounces	170	ml.
4 fluid ounces	114	ml.
3 fluid ounces	85	ml.
2 fluid ounces	57	ml.
1 fluid ounce	28·4	ml. (Approx. 30 ml.)
60 ℥ (minims)	4	ml.
50 ℥	3	ml.
45 ℥	2·7	ml.
30 ℥	1·8	ml.
20 ℥	1·2	ml.
15 ℥	0·9	ml. (Approx. 1 ml.)
10 ℥	0·6	ml.
8 ℥	0·5	ml.
5 ℥	0·3	ml.
3 ℥	0·18	ml.
2 ℥	0·12	ml.
1 ℥	0·06	ml.

MILLIEQUIVALENTS

Chemical substances in body fluid react together not by their absolute weights but by their equivalent weights. (The equivalent weight of an element is that weight which will combine with or take the place of 8 parts by weight of oxygen. For univalent elements the equivalent weight is the same as the atomic weight; for divalent elements it is half the atomic weight and so on). It is convenient to express in terms of milliequivalents substances, such as acids and bases, which react together. Acid radicles are chloride, bicarbonate (expressed in terms of CO_2), phosphate, sulphate, organic acids and proteins. Basic radicles are sodium, potassium, calcium and magnesium. A milliequivalent of any substance is the amount contained in 1 ml. of its normal solution. (A normal solution contains the equivalent weight of a substance in grammes per litre. The term "normal saline" however is physiological or isotonic saline and is only about one-sixth the strength of chemically normal saline). 1 milliequivalent of sodium is 23 mg. and so on.

The term milliequivalent per litre (mEq/litre) means the thousandth part of the equivalent weight of the substance in grammes per litre. The simplest way of converting mg. per 100 ml. into mEq/litre is to express the substance as mg. per litre and divide by its equivalent weight (=atomic weight ÷ valency), i.e.,

$$\text{mEq/litre} = \frac{\text{mg. per 100 ml.} \times 10}{\text{equivalent weight.}}$$

Conversely, mEq/litre can be converted into mg. per 100 ml. by multiplying mEq/litre by the equivalent weight and dividing by 10,

$$\text{i.e., mg. per 100 ml.} = \frac{\text{mEq/litre} \times \text{equivalent weight.}}{10}$$

The cases of bicarbonate and proteins require special explanation.

Bicarbonate.—We measure the plasma bicarbonate by the amount of CO_2 which it will yield—normally about 60 ml. per 100 ml. of plasma or 600 ml. per litre. Now the molecular weight in grammes of any gas occupies 22.4 litres at normal temperature and pressure. In the case of CO_2 the molecular weight is also the equivalent weight. If 600 is divided by 22.4 we get 27 which is the figure for the mEq/litre of CO_2 derived from bicarbonate.

Proteins.—Proteins act as weak acids and are expressed in terms of the amount of base they neutralise. The average equivalent weight of proteins is that part of their molecular weight which will neutralise 1 equivalent of NaOH. When the concentration of protein in mg. per litre is divided by this we obtain a figure for the milliequivalents of protein. It is more simple to use the formula:

$$\text{Protein in G. per 100 ml.} \times 2.43 = \text{mEq/litre.}$$

Blood plasma has normally about 7 G. protein per 100 ml. and thus has the power of binding about 16 mEq/litre of base.

Equivalent weights of common substances are:—sodium 23; potassium 40; calcium 20; chlorine 35.5; sodium chloride 58.5; bicarbonate 22.4. (The latter is not strictly the equivalent weight, but as explained above, is used in the same way).

IDENTIFICATION OF TABLETS

Many doctors prescribe tablets which they rarely see and with whose appearance they are unfamiliar. Loose tablets have sometimes to be identified in an emergency and so a study of what various tablets look like is worth while. Although the addition of

colouring to B.P. tablets other than those specified is not official and pharmacists generally disapprove of recognising tablets by colour, the doctor, for his own convenience, may consider it advisable to select for his bag brands of tablets of distinctive marking and colour so as to lessen any chance of confusion in an emergency. Some tablets, such as Gantrisin, Tromexan, Stovarsol and Disprin have their full name impressed on them. The distinctive markings (usually an abbreviation of the chemical name or the maker's name) of some others are as follows:—Sulphamezathine—SZ; Sulphadiazine—SDZ; Sulphamerazine—SMZ; Mepacrine—Q; Dilaudid (and other Knoll tablets)—K.

THE USE OF THE TELEPHONE IN EMERGENCIES

In order to call the police or an ambulance the telephone should be used in accordance with the instructions issued by the Post Office. With the automatic (dial) telephones of London and certain other places, 999 should be dialled. This does not call the police or ambulance direct but makes a special signal at the telephone exchange. The call is then passed to the appropriate emergency authority by the operator according to the request of the caller. In some areas emergency calls are made by dialling "0" or "01" or by pressing an emergency button.

EXTRACT FROM RULES FOR THE BROADCASTING OF S.O.S. AND SIMILAR MESSAGES

1. For relatives of sick persons.

The British Broadcasting Corporation (Broadcasting House, London, W.1., Telephone LANgham 4468) will broadcast messages requesting relatives to go to a sick person only when the hospital authority or the medical attendant certifies that the patient is *dangerously ill*, and if all other means of communication have failed. In the normal course of events messages will be broadcast only when the full name of the person wanted is available.

NOTE.—When the person sought is known to be on board a ship at sea, a message can only be broadcast if the ship is not equipped with apparatus for the reception of messages by wireless telegraphy. Further, there must be a possibility that the return of the person sought can be hastened by the reception of such a message. This is not considered to be so when the ship

is on its way to a known port. In such cases, enquirers are advised to communicate with the owners or agents of the ship or with the port authorities.

In no case can an S.O.S. be broadcast requesting the attendance of relatives *after death has occurred*.

2. Appeals for special apparatus, foods or medicines.

These appeals will be broadcast only at the request of major hospitals, and after every other means of obtaining the required item has failed.

No message can be broadcast regarding lost animals or property, except where there is real danger to life, as from the theft of dangerous drugs or from escaped wild animals, and then only at the request of the police.*

There is no charge for broadcasting S.O.S. messages.

INFECTIOUS DISEASES IN GENERAL WARDS

The occurrence of an infectious fever in a general ward creates an urgent situation for the doctor in that action and advice are needed at once. The following notes are intended to guide him.

There is no rule of thumb plan about isolation and quarantine. Our aim should be to prevent spread of the disease to susceptible patients, and at the same time, not to interrupt the work of the ward unnecessarily. Surveillance often achieves this object better than quarantine, but the nursing staff should be told what to look for, *e.g.*, slight coryza and rise of temperature. Measles, rubella, chicken-pox and mumps can be regarded as almost inevitable and it is a good thing if healthy children can get over them before they grow up. Elaborate measures to escape them are not to be encouraged except in special cases. Any members of the staff who have not had the disease in question should be carefully watched. It is best to let them leave the children's or maternity ward for an adult ward.

Erysipelas, meningitis and poliomyelitis are of low infectivity and patients can be nursed in general wards, if otherwise convenient, by barrier methods. In order to obtain special treatment and for administrative reasons it may be wiser to transfer them.

In the case of certain diseases the Medical Officer of Health must be notified. In some areas he wishes to know about rubella

*If a doctor loses his bag he should emphasise the fact that it contained dangerous drugs. This will expedite measures to recover it.

also and other diseases may be made notifiable in special circumstances, *e.g.*, chicken-pox during an epidemic of smallpox.

The following is a list of notifiable infectious diseases in England, Wales, Scotland and Northern Ireland:—

Cholera.	Pneumonia, Acute Influenzal.
Diphtheria.	Poliomyelitis, Acute.
Dysentery.	Psittacosis (N. Ireland only).
Encephalitis (Acute) (Scotland	Puerperal Pyrexia.
Infective N. Ireland Leth-	Relapsing Fever.
argica).	Rheumatic Fever (N. Ireland
Enteric (Typhoid and Para-	only).
typhoid) Fever.	Scarlatina or Scarlet Fever.
Erysipelas.	Smallpox.
Gastro-enteritis in children under	Trachoma (N. Ireland only).
two years (N. Ireland only).	Tuberculosis (N. Ireland to
Malaria.	Tuberculosis authority).
Measles (not in Scotland).	Typhus.
Membranous croup.	Undulant Fever (N. Ireland
Meningococcal Infection (Scot-	only).
land and N. Ireland—Cerebro-	Vincent's Angina (N. Ireland
spinal Fever).	only).
Ophthalmia Neonatorum.	Whooping Cough.
Yellow Fever (N. Ireland only).	Food poisoning (or suspected
Pemphigus (N. Ireland only).	„ food poisoning) (not in Scot-
Plague.	land; N. Ireland Bacterial
Pneumonia, Acute Primary.	only).

Measles.—A child who contracts measles should be sent home or to the isolation hospital. If this is impracticable isolate him in a separate room and use barrier nursing techniques. Find out which of the other children have had measles. (This should be stated on the admission sheet but it is as well to verify the evidence). Nothing further need be done about these children. Give the parents of children who are well enough and who have not had measles the option of taking them home, possibly to develop this inevitable disease.

Gamma globulin (obtainable from The Central Public Health Laboratory, Colindale Avenue, London N.W.9. Telephone COLindale 7041) may be used to prevent or modify an attack. It is difficult and expensive to prepare and its use should be confined to children who have some other serious disease or who are “weakly.” If the supply is short restrict its use to children in beds next to the measles case rather than give a smaller dose all round. As the attack rate in infant contacts under six months is low they need not be given gamma globulin if one can be quite sure that the mother has had measles.

Gamma globulin is issued in ampoules containing 250 mg. of protein which should be dissolved in 3 ml. of sterile distilled water. It is given subcutaneously or intramuscularly within two or three days of the rash in the infecting case in doses according to the following table. Immunity conferred by gamma globulin lasts from three to four weeks.

	<i>Under 1 year</i>	<i>Over 1 year and under 3 years</i>	<i>Over 3 years</i>
Prevention . .	3 ml. (250 mg.)	6 ml. (500 mg.)	9 ml. (750 mg.)
Modification .	3 ml. (250 mg.)	3 ml. (250 mg.)	3 ml. (250 mg.)

If gamma globulin is unobtainable convalescent serum (preferably from a single donor) may be used. It should be given from the first to fifth day after exposure in doses of 0.3 ml. per pound (0.6 ml. per Kg. approx.) body weight (for prevention) or 0.1 ml. per pound (0.2 ml. per Kg. approx.) body weight (for modification). The use of pooled adult serum should be abandoned because of the risk of serum hepatitis. If preventive doses are given, admissions and discharges can go on as before.

Mumps.—The primary case should be sent home or to the isolation hospital. Nothing need be done for fourteen days and then, if there are susceptible children left, the ward should be closed for a week. The children should be watched for early symptoms. The likelihood of cases developing after the twenty-first day is very slight. Only children who are definitely known to have had mumps should be admitted during the period of surveillance.

Rubella.—This is not highly infectious and secondary cases are not inevitable. Hence when the primary case has been disposed of, surveillance only should be practised. A child should not be sent home if his mother is in the early stages of pregnancy (*i.e.*, up to the end of the fourth month). If there has been contact the mother should receive 750 mg. of gamma globulin.

Diphtheria.—The patient should be isolated—usually in the infectious diseases hospital. All contacts (patients and staff) should be questioned about immunisation and their throats and noses should be swabbed. Those who are positive should be isolated, and the others should be carefully watched. Un-immunised children and staff with negative swabs may be given passive immunity (which lasts fourteen to twenty-one days) by antitoxin (2,000 to 4,000 units intramuscularly). Active immunisation should also be started.

Scarlet fever.—The case should be sent home, or to the isolation hospital or to a separate room and treated with penicillin. Scarlet fever is usually mild and uncomplicated but it should not be regarded lightly in a surgical or maternity ward. Throat swabs should be taken from the remaining patients and staff and those who show hæmolytic streptococci or who have symptoms should be isolated and given penicillin. This eliminates the streptococci in four or five days and drug resistance does not develop. The ward need not be closed for admissions because of a single case but closure may be necessary for a few days if further cases occur.

Chicken-pox.—The child should be sent home or to an isolation hospital. Chicken-pox is very infectious and as cases are not usually diagnosed in the early stages secondary cases can be expected. A history of chicken-pox should be regarded as doubtful if careful search does not show any pock marks. If susceptible children are left in the ward only patients who give a definite past history of chicken-pox should be admitted for twenty-one days—the quarantine period. An adult with herpes zoster may give rise to chicken-pox in child contacts.

Smallpox.—If this is suspected ring up the Medical Officer of Health and act on his instructions.

Whooping cough.—The case (suspected because of a spasmodic cough and a lymphocytosis or proved by a per-nasal swab), should be sent home or to the isolation hospital. Further cases will not occur until the end of the incubation period—say in seventeen days. Hence admission of children with a definite past history of whooping cough can go on for the first ten days. Children in the ward who have not had whooping cough may be given gamma globulin as a prophylactic in doses double those recommended for measles. Admissions should cease from the tenth to twenty-first

day after the case has been received and the remaining children should be carefully watched.

Sonne dysentery.—The patient should be sent to the isolation hospital or isolated under strict barrier nursing precautions until several consecutive stool cultures are negative. Rectal swabs, or preferably stools, of all contacts should be examined. Members of the staff must report any diarrhoea. Admissions need not be restricted.

	INCUBATION in days	INFECTIVITY	QUARANTINE in days
Chicken-pox	Extremes 10 to 16 Commonest 14	Until all scabs are off	21
Diphtheria	2 to 10	Until two consecutive nose and throat swabs are negative	7
Measles	7 to 14 Normally Koplik's spots on the 9th day and rash on the 12th day	14 days from the appearance of the rash or, in mild attacks, until the end of clinical symptoms	21
Rubella	5 to 21	7 days from the appearance of the rash	21
Mumps	Extremes 10 to 28 Commonest 17	1 week after glands subside	28
Whooping cough	14 to 16	21 days or until all catarrh has gone	14

FIG. 69

Incubation, Infectivity and Quarantine.

EMERGENCY TRAVEL ABROAD

Most invalids can be accepted for air travel, but may be refused, unless accompanied by a doctor or nurse. The cost of this, and possibly that of displacing additional seats to accommodate a stretcher, may deter the traveller. The St. John Ambulance Brigade can provide Air Attendants.* Doctors requiring advice regarding the carriage of invalids should consult the Medical Department of the air line concerned (*see also page 398*). Health certificates are a commoner cause of trouble.

Although most countries are now signatories to the International Sanitary Regulations (1951), of the World Health Organisation, the requirements of certain foreign governments for certificates of immunisation may vary and are apt to be changed. Travellers, particularly to the East, should therefore consult the Traffic Departments of the air line or shipping companies, accredited travel agents, or the Embassy or Consulate of the country concerned. The following notes are provided to help any doctor who is asked to advise on health certificates by someone suddenly called abroad. They apply to all forms of travel whether by land, sea or air.

In addition to the specific requirements of particular countries, all persons going overseas are advised to be effectively inoculated against typhoid and paratyphoid. For typhoid and any other disease, for which there is no international certificate, an ordinary certificate from any doctor is sufficient.

International certificates of vaccination are now only necessary in the case of smallpox, yellow fever and cholera. In the case of vaccination against smallpox, if the first attempt at primary vaccination fails, at least two more attempts with different batches of lymph should be made, and the results recorded (*see page 611*).

Any doctor can vaccinate against smallpox and cholera and certify, but yellow fever inoculation and certification can only be carried out at special inoculation centres (*see page 635*). British European Airways Medical Services at Northolt, Renfrew and London Airport can provide inoculation against smallpox and

* For these apply on Monday to Friday, from 9.30 a.m. to 5.30 p.m. to Officer in charge of Register, St. John Ambulance Brigade Headquarters, 8 Grosvenor Crescent, London, S.W.1. Tel. SLOane 9861, and on Saturday and Sunday, 8 a.m. to 10 p.m. to Officer in charge of Register, St. John House, 15/16 Collingham Gardens, London, S.W.5. Tel. FRObisher 6477.

typhoid but not against yellow fever. Blank forms of the international certificate against smallpox or cholera must be obtained by the traveller from the transport company, or from the Ministry of Health, 23 Savile Row, London, W.1. (Tel. REGent 8411), or the Department of Health for Scotland, St. Andrew's House, Edinburgh, 1 (Tel. WAVErley 7241). It is essential to have the doctor's signature on any international certificate (other than a yellow fever certificate), authenticated by the local Medical Officer of Health. Certificates of inoculation against smallpox and cholera by practitioners resident in Eire will be authenticated if forwarded to the Department of Health, Customs House, Dublin.

Occasional difficulty arises when the person wishes to travel urgently by air and has only just been vaccinated for the first time, so that his certificate is not yet valid. The airline might accept him as a passenger, if he completes a form of indemnity against liability for delays, expenses, etc. He would, however, have to take the risk of being refused admission or being detained in quarantine at his destination, though he might get in on compassionate grounds. Emergency travel would probably not be by sea and in any case would allow time for vaccination by the ship's surgeon.

The main facts about immunisation are given in Figure 70. Additional points are (1) The periods of validity may vary in countries not bound by the International Sanitary Regulations. (2) The minimum interval between doses of T.A.B.C., typhus and plague vaccines is seven days. (3) Parents consent for inoculation is necessary in the case of children under 18 years of age.

Sequence of Inoculations

Inoculations may have to be given as quickly as possible in order to allow certificates to become valid before departure. The following sequence practised by B.O.A.C. is convenient.

Day	Vaccine
1st	Anti-yellow fever (0.5 ml.). Anti-cholera (1 ml. of 8,000 million vibrios). Anti T.A.B.C. (1st dose—0.25 ml.).
4th	Vaccine-lymph against smallpox.

GUIDE TO IMMUNISATION FOR INTERNATIONAL TRAVEL

(Sir Harold Whittingham)

Immunisation against	Immunising Agent	Lower Age Limit	Dosage of Vaccine		Validity Period of Certificate	Booster Dose of Vaccine	Remarks
			Adults and Teenagers	Children			
(1)	(2)	(3)	(4)	1-5 6-12 (5) (6)	(7)	(8)	(9)
SMALLPOX	Vaccine lymph	None	(a) 10 multiple pressures		(a) 8 days to 3 years from date of primary vaccination	Re-vaccination every 3 years by 30 multiple pressures	(a) Primary vaccination should be subsequent to yellow fever inoculation (b) Re-vaccination should normally be done 4 days after yellow fever inoculation, but, if done first, must precede yellow fever inoculation by 7 days
	(b) Re-vaccination		(b) 30 multiple pressures		(b) For 3 years from date of vaccination		
YELLOW FEVER	Attenuated strain of pantropic virus 17D vaccine	None	0.5 ml.	0.5 ml	10-12* days to 6 years from date of inoculation If re-inoculated within the previous validity period, becomes valid at once for 6 years, otherwise only after 10-12 days	0.5 ml. every 6 years	Should precede primary vaccination by at least 4 days. If given subsequent to primary vaccination there must be an interval of 21 days
	1,000 Mouse units per ml.						
CHOLERA	Vibrio cholerae vaccine 8,000 millions per ml.	1 year	1.0 ml.	0.2 ml	6 days to 6 months from date of inoculation	1.0 ml. 6-monthly with appropriate body weight dose for children	

Immunisation against	Immunising Agent	Lower Age Limit	Dosage of Vaccine			Validity period of Certificate	Booster Dose of Vaccine	Remarks
			Adults and Teenagers	Children				
(1)	(2)	(3)	(4)	1-5	6-12	(7)	(8)	(9)
TYPHOID	(i) Alcoholised T.A.B.C. vaccine containing	1 year	Dose : (1st) 0.25 ml. (2nd) 0.5 ml.	0.1 ml. 0.2 ml.	0.2 ml. 0.4 ml.	Not an international requirement	0.25 ml. yearly	Is a recommendation only
	A. 1,000 millions per ml. B. 500 millions per ml. C. 500 millions per ml. (ii) Non-alcoholised vaccine of similar number of organisms							
PLAGUE	Pasteurella pestis vaccine 2000 millions per ml.	1 year	Dose : (1st) 0.5 ml. (2nd) 1.0 ml.	0.1 ml. 0.2 ml.	0.2 ml. 0.4 ml.	Not an international requirement	1 ml. yearly with appropriate body weight dose for children	Only done where it is a specific requirement by the country of destination or transit. Is not a normal international requirement
TYPHUS	Epidemic and murine virus vaccine cultured in yolk-sac	1 year	Dose : (1st) 1.0 ml. (2nd) 1.0 ml.	0.2 ml. 0.2 ml.	0.4 ml. 0.4 ml.	Not an international requirement	1 ml. yearly with appropriate body weight dose for children	Only done where it is a specific requirement by the country of destination or transit. Is not a normal international requirement

* In Ceylon, India and Pakistan the validity period for yellow fever is 12 days to 6 years.

FIG. 70

7th - 9th Anti T.A.B.C. (2nd dose—0.5 ml.), also read results of primary vaccination against smallpox and enter on the International Certificate whether it is “ successful ” or not. The result of re-vaccination against smallpox has not got to be recorded.

Anti-yellow fever vaccination should always precede smallpox vaccinations by four days as, if smallpox vaccination was done first, 21 days would have to elapse before giving yellow fever vaccine. Smallpox and yellow fever inoculations should never be done at the same time. Yellow fever inoculation does not cause any reaction.

A passenger is liable to be vaccinated compulsorily or detained in quarantine if his certificates are not in order. Conscientious objectors are treated similarly. If for medical reasons the doctor decides that inoculations are inadvisable he should certify this, but his certificate is no guarantee against the passenger being quarantined.

LUMBAR PUNCTURE

(For hazards see page 21)

Position.

The patient should lie on his left side with his buttocks and shoulders on the hard edge of the bed (Fig. 71). If the mattress sags, put fracture boards under it. The long axis of the spine should be horizontal, and the plane of the iliac crests vertical. The spine must be fully flexed and the patient should be asked to get the chin as near to the knees as possible. A roller towel placed round the neck and knees, and tightened by twisting with a rod sometimes helps to obtain and maintain the flexed position.

Site of Puncture.

The usual site is the space between the spines of the 3rd and 4th lumbar vertebræ (*see page 21*). This space is on a plane passing through the highest points of both iliac crests.

The Puncture.

If difficulty is expected a stout (Barker) needle, or a nickel needle (*see page 16*) should be used. For routine use a smaller needle is better. Punctures must be made under full aseptic precautions. A finger-grip in front of the orifice, as in Dattner's

needle (Fig. 74, page 525) avoids the need of holding and perhaps contaminating, the shaft. Everything should be dry including the operator's hands. Gloves are not essential and instead of them a sterile towel may be used through which, the needle is held and the skin palpated.



FIG. 71

Position for lumbar puncture. The nurse places one arm under the knees and the other round the back of the neck in order to flex the spine as fully as possible.

A wheal of 2 per cent. procaine is raised in the skin over the junction of the lower and middle thirds of the interspace. The skin is pierced here by giving the lumbar puncture needle a rolling motion. The direction of the needle is then readjusted so that while in the horizontal plane it is inclined about 5 degrees towards the head. It is then parallel to the slope of the vertebral spines. No resistance is felt until the ligamentum flavum is reached. This is pierced with the bevel in the spinal axis (see page

22). The dura is next encountered and penetrated. Withdrawal of the stylet allows C.S.F. to flow.

Difficulties.

- (1) No fluid flows. Advance the needle a few millimetres and rotate it in case a nerve root is obstructing it.

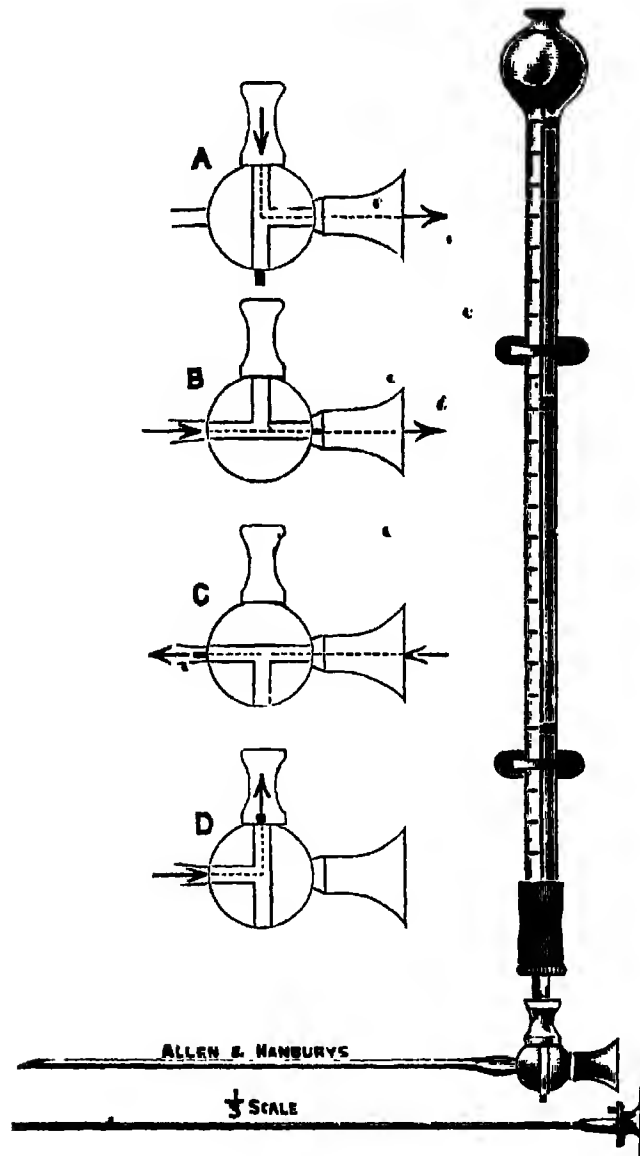


FIG. 72

Greenfield's spinal manometer.

- (2) Bone is encountered. This usually means that flexion is incomplete. Withdraw the needle and adjust the position.
- (3) Blood appears. If it is only a few drops and then nothing more, it means that the needle has not gone far enough but is in the subdural space. By over enthusiastic insertion, one may encounter the anterior subdural space.

If the fluid is bloodstained the decision must be made as to whether the "bloody tap" is due to trauma or subarachnoid hæmorrhage. Traumatic bleeding often clears as the fluid drains.

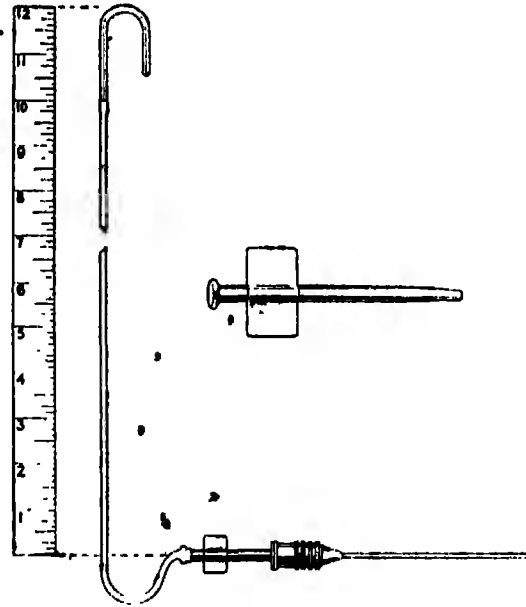


FIG. 73
Northfield's apparatus for measuring the cerebro-spinal fluid pressure.

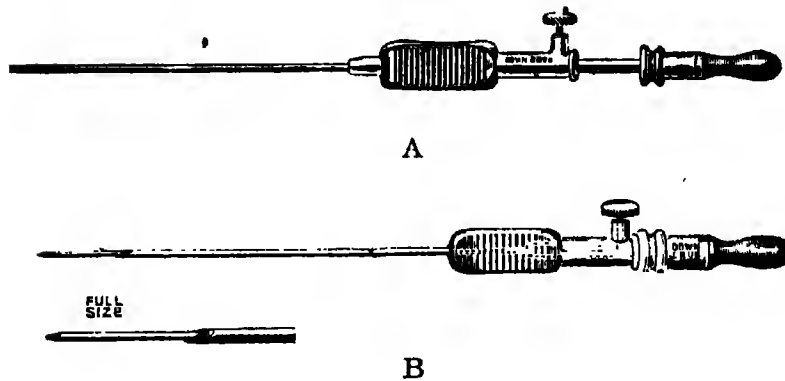


FIG. 74
Dattner's needle.

- A Arranged for introduction;
- B With needle projecting after introduction.

If in doubt, take specimens in three numbered tubes and have red cell counts done on all three. A diminishing count indicates traumatic blood. Clotting in the fluid may occur if bleeding was due to trauma, and if centrifugalised the supernatant fluid is colourless. In subarachnoid hæmorrhage clotting does not occur and the supernatant fluid is yellow.

Measuring the C.S.F. pressure.

This cannot be done by observing the rate of flow. A manometer, such as Greenfield's (Fig. 72) must be attached to the needle. A simpler method is to insert into the needle a butterfly adaptor with a length of bicycle valve tubing attached and at the other end of which is a small glass "U" tube. This is held below the level of the needle until fluid appears in it, and then raised until the fluid in one of its limbs is steady. Since CO_2 retention raises C.S.F. pressure it is advisable to wait until breathing is normal. A metal ruler with its zero mark level with the needle serves to read the pressure (Fig. 73).

Note the resting pressure and its response to coughing and straining. Failure to respond means that the needle is not in the correct place (or that there is complete block). Compress the right and left internal jugular veins separately and then together and record results (Queckenstedt's test).

The Dattner (double) needle.

This is useful if lumbar puncture has to be performed in cases of raised intracranial tension, and also to minimise the risk of post-puncture headache.

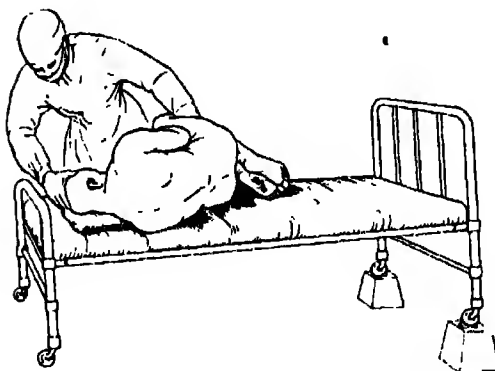


FIG. 75

Position for cisternal puncture in a comatose patient.

It (Fig. 74) consists of a fine needle and stylet inside a larger needle. The outer needle is inserted down to the ligamentum flavum, and the inner needle punctures the dura. If used carelessly the outer needle may penetrate the dura and the advantage of Dattner's principle will be lost. If correctly performed, fluid should cease to flow when the inner needle is with-

drawn into the outer case. A syringe must be used to withdraw C.S.F. Manometry is not possible.

CISTERNAL PUNCTURE

(For hazards see page 22)

Position.

The best position for a comatose patient is lying on the left side with his head at the foot of the bed so that the bedpost is

not in the way. The head is supported on a small sandbag and flexed. The spine should be horizontal (Fig. 75). The sitting position may be used in the conscious patient.

Site of puncture.

This is determined by the fact that a horizontal plane through the tips of the mastoid processes bisects the atlanto-occipital space

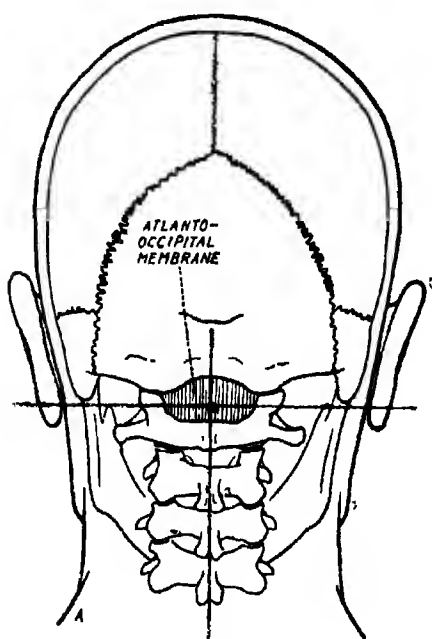


FIG. 76A

The point of entry of the needle at the intersection of a line joining the tips of the mastoids and the vertical midline of the neck.

(*"Pye's Surgical Handicraft."*)

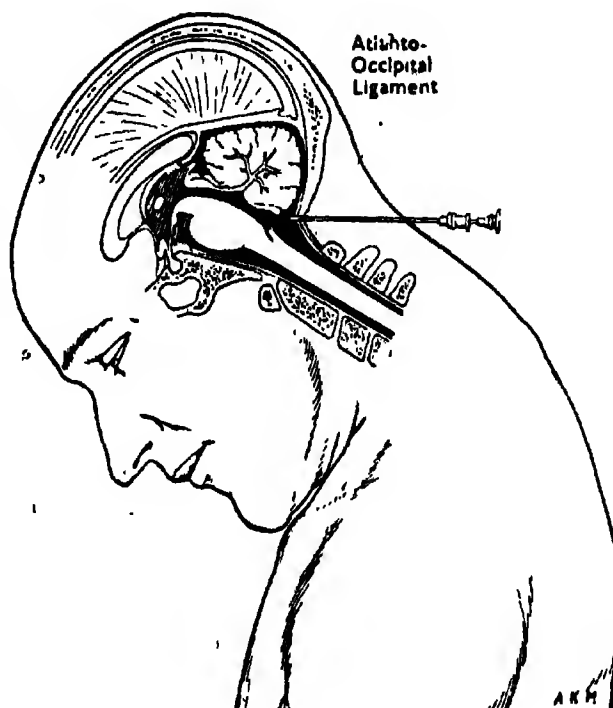


FIG. 76B

Showing the direction of the needle which has reached the atlanto-occipital ligament.

(*Surgery of Modern Warfare*)

(Fig. 76A). The tips of the mastoid processes are marked, and a horizontal line joining them is drawn using a tape measure and a grease pencil. The point where this line crosses the vertical midline of the neck is marked. This is the entry point.

The puncture.

A Purves-Stewart graduated needle (Fig. 77) is convenient but a lumbar puncture needle may be used. A scratch made on it 5 cm. from the tip is a useful guide.

The needle is directed slightly upwards so that the occipital bone is hit (Fig. 76B). It is then worked down tapping the bone periodically until the atlanto-occipital ligament is reached. When this is pierced there is a characteristic "give." The needle is then

advanced cautiously, removing the stylét at intervals until fluid appears. Some operators prefer to mount the needle on a syringe and apply slight suction when the atlanto-occipital ligament is pierced.

A rough idea of the depth at which the ligament will be reached is given by the neck circumference at the level of the

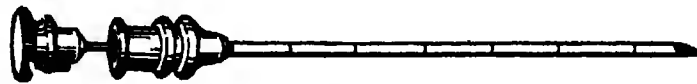


FIG. 77

Purves-Stewart's cisternal puncture needle.
(*"Pye's Surgical Handicraft."*)

upper border of the thyroid cartilage divided by nine. In an adult the cistern is about 2 inches from the skin and the medulla is about half an inch further on.

STELLATE GANGLION BLOCK

The object is to infiltrate with local anæsthetic the region of the stellate ganglion on the side of the affected cerebral hemisphere (*i.e.*, the opposite side to the paralysed arm and leg). The anterior route is the safest since the needle is above the dome of the pleura and there is little danger of pneumothorax. The injection is aimed at the upper part of the ganglion and we rely on procaine seeping down to the part of the ganglion which overlies the neck of the first rib. The entry point for the needle is a centimetre below the level of the lower border of the body of the 6th cervical vertebra close to the lateral border of the trachea. Two landmarks indicate the 6th cervical vertebra. The cricoid cartilage lies opposite the body and the large anterior tubercle of its transverse process (Chassaignac's tubercle) is fairly easy to feel. The entry point can also be marked from below being about 4 cm. above the inner end of the clavicle at the median border of the sterno-mastoid muscle. Raise a wheal of 1 per cent. procaine at this point. Press the left middle finger deeply on the inner border of the sterno-mastoid muscle and palpate and displace laterally the carotid artery. Insert a 4-inch needle along the finger and through the wheal straight backwards between the carotid artery and the trachea until bone (the body of the seventh cervical vertebra) is felt. These relationships are shown in Figure 78. The stellate ganglion does not lie on the periosteum but on the

longus colli muscle and so the needle should be withdrawn 0.5 to 1.0 cm. and a negative aspiration test obtained before injecting 5 ml. of 1 per cent. procaine. After withdrawing the needle a further 1 cm. 5 ml. more are injected. The patient should be told to resist the desire to cough. Pain may be felt in the chest and arm but will pass off when the procaine is injected.

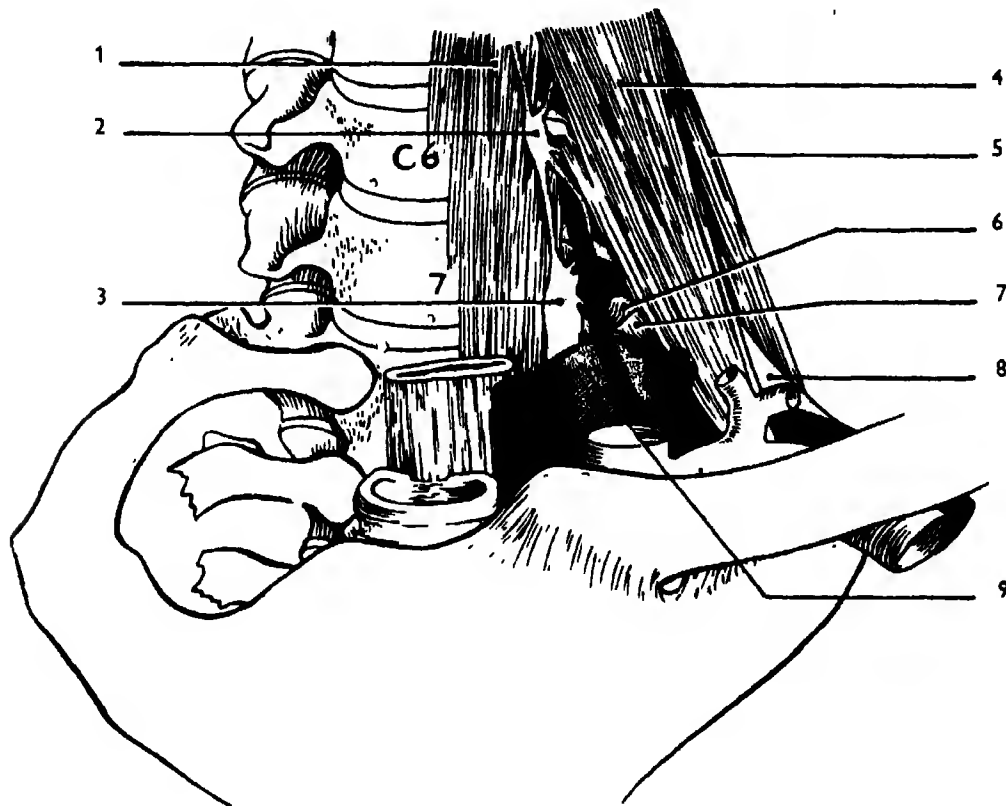


FIG. 78

The relations of the stellate ganglion.

1. Longus colli muscle. 2. Middle cervical ganglion. 3. Stellate ganglion. 4. Scalenus anterior muscle. 5. Scalenus medius muscle. 6. Transverse process of first thoracic vertebra. 7. Tubercle of first rib. 8. Brachial plexus. 9. Dome of Pleura.

A successful injection is followed in about 10 minutes by Horner's syndrome (ptosis, miosis, enophthalmos, flushing of the face and conjunctiva and possibly unilateral blocking of the nose from engorgement). When performed for cerebral thrombosis or embolism the injection should be repeated every six hours during the first day and then daily for up to 15 days according to progress. It is possible to thread a fine polythene tube down the needle and to leave it in place for subsequent injections.

PARACENTESIS OF THE PERICARDIUM

There are three possible routes—anterior, epigastric and posterior.

- (1) **Anterior.** The needle is inserted (a) in the fifth left interspace just outside the apex beat but inside the outer edge of the cardiac dulness, or (b) in the fourth left interspace one inch from the sternal margin (to avoid the internal mammary artery). The right side should be avoided lest the right auricle be injured.

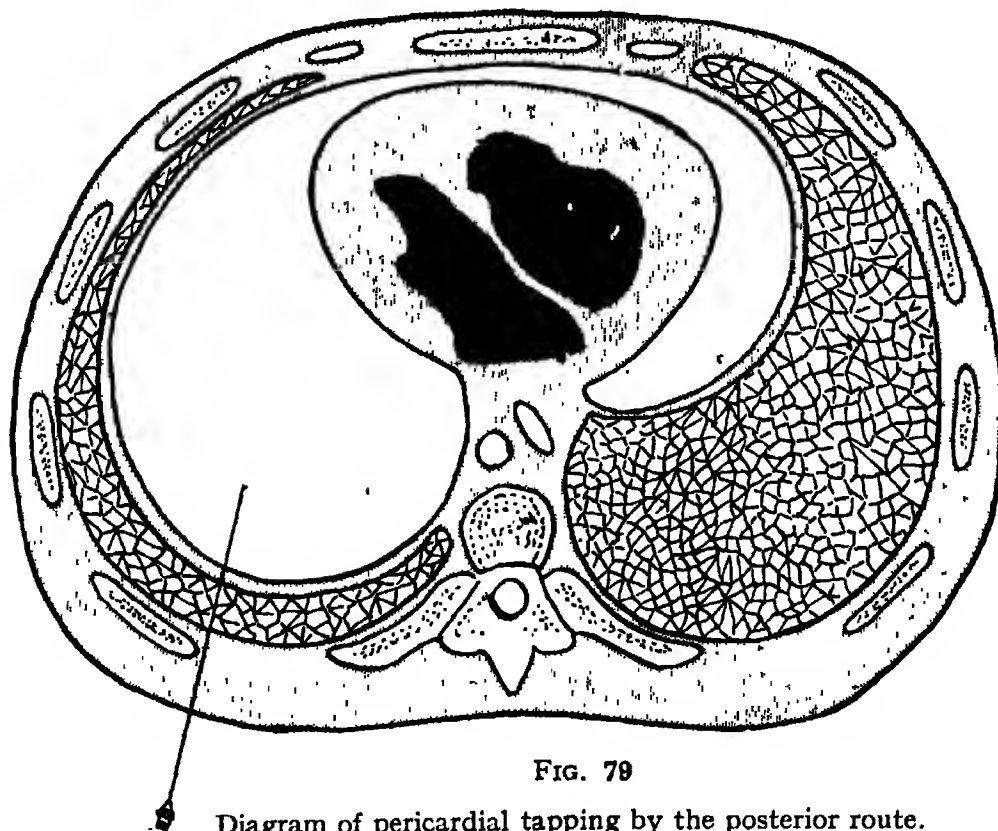


Diagram of pericardial tapping by the posterior route.

- (2) **Epigastric.** The needle is inserted at the costo-xiphoid notch and directed upwards and backwards to enter the lowest part of the pericardial sac.
- (3) **Posterior.** The needle is inserted near the inferior angle of the scapula on the left side. It traverses the lung to reach the pericardium (Fig. 79).

The skin is cleansed, and the skin and subcutaneous tissues infiltrated with 2 per cent. procaine. A long needle of about 1 mm. bore is used for the pericardial tap. The anterior routes are safer than the posterior, but it is not possible to remove large

quantities of fluid by them. When an effusion is very large it collects posteriorly and compresses the lung (Fig. 79). In such a case the posterior route is best. When empyema of the pericardium is suspected, approach through the pleura and lung is contraindicated and the epigastric route, using a fine trocar and cannula, is to be preferred. It is probably wise in such a case to enlist the help of a surgeon.

ARTIFICIAL PNEUMOTHORAX INDUCTION

In young or sensitive patients sedation is advisable and Linctus of Codeine B.P.C. should be given if there is a cough.

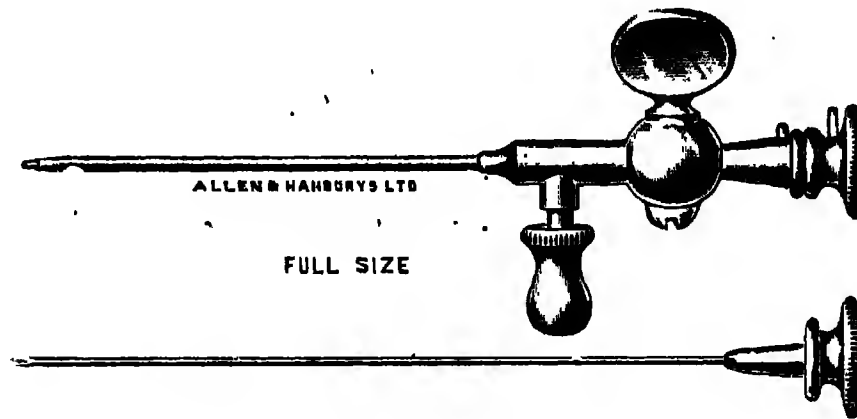


FIG. 80
Kuss Needle.

The patient lies on the "good side" with his upper arm raised to expose the axilla. With aseptic precautions the skin and tissues down to the pleura are infiltrated with 2 per cent. procaine in the fourth, fifth or sixth inter-costal spaces in the mid-axillary line.

A needle with a flat end and sharp trocar such as the Kuss needle (Fig. 80) attached to an artificial pneumothorax apparatus and open to the manometer is inserted down to the pleura. It is best to choose a spot at the upper border of a rib so as to avoid the vessels in the sub-costal groove of the rib above. The sharp trocar is then removed. Very often a sharp "hiss" as air rushes in shows that the pleura has been entered. If it has not, the blunt trocar should replace the sharp one before further insertion. A good negative swing of the manometer fluid in both phases of respiration indicates that the needle is in the pleural space. The

clips are then turned on and air is drawn into the chest. The usual amount for induction is 100 to 300 ml.

Failure to enter the pleural space is shown by the behaviour of the manometer:—

- (1) A small equal swing on each side of zero means that the needle is in the lung tissue. A similar but larger swing indicates that it is in a lung cavity.
- (2) A rising positive pressure means that the needle has punctured a blood vessel. It should be withdrawn promptly.
- (3) Absence of any reading means that the point of the needle is in the chest wall, fluid, or an adhesion, or that the tubing or needle is blocked. But a small negative swing of the order of -1 -3 is sometimes obtained when the needle point is situated extra-pleurally.

In the case of failure, other sites should be tried before abandoning the procedure, such as the 7th or 8th spaces posteriorly, and the 2nd, 3rd, and 4th spaces anteriorly.

Numerous types of apparatus exist, the earlier ones consisting simply of two bottles and a manometer. When using an apparatus of this type for an induction, the fluid levels in each bottle should be at the same height. The negative intra-pleural pressure draws air into the chest. If the fluid levels are different, air enters the chest under pressure and the risk of air embolism is increased. In some machines the manometer is open to the needle all the time. In others a tap or clip has to be opened but with all, the pressure changes must be carefully observed.

PNEUMO-PERITONEUM INDUCTION

The most convenient site for the intra-peritoneal injection of air is near the left subcostal margin just external to the rectus abdominis muscle.

With aseptic precautions a wheal of 2 per cent. procaine is raised in the skin with a small hypodermic needle. The syringe (2 ml.) is recharged and, using a long serum needle, the abdominal wall is infiltrated down to the peritoneum. When this is punctured the sense of resistance to the piston disappears. Sudden penetration should be avoided. The piston should be withdrawn to make sure that the needle point is not in a blood vessel. A pneumothorax apparatus is then attached to the needle by an adaptor. Air is slowly injected in amounts up to 1,000 ml.

A manometer is unnecessary, but if available provides a useful indication that the needle is in the correct space by showing respiratory excursions and also a rise when the abdomen is pressed on. If before the air is run in the clips are suddenly opened and closed, the fluid in the manometer will rise and fall rapidly to almost zero if the peritoneal space has been entered. If the needle is outside the peritoneum, the pressure will remain high.

Some operators prefer a pneumothorax needle,* and others a special Veress needle. This consists of a sharp needle in which runs a hollow, blunt-ended trocar with a lateral hole. The trocar is made to project slightly from the needle by a spring, thus ensuring that the gut is pushed away by the blunt end rather than the sharp needle point.

Clinical evidence that air has entered the peritoneal space is provided by:—

- (1) Pain in the shoulder regions (but not on the side on which the phrenic nerve has been interrupted).
- (2) Disappearance of liver dullness.

The most frequent cause of failure to induce a pneumoperitoneum is that the needle is not inserted far enough.

CONTINUOUS SUCTION OF PNEUMOTHORAX

(For indications see page 137)

Several methods are available.

(1) **Sprengel's pump using water.**

The patient's bed is moved near to a water tap to which a Sprengel's filter pump is attached. This is connected to a needle in the chest (Fig. 16, page 138). A water bottle should intervene to indicate that air is being aspirated and a manometer should be connected to a side arm of the needle.

(2) **Sprengel's pump using oxygen.**

Using a filter pump with a very fine jet, a similar apparatus can be set up using oxygen instead of water (Fig. 81). It is wise to protect the outlet of the pump by wire gauze.* A flow of 4 litres per minute will produce a negative pressure of 8 ml. of water.

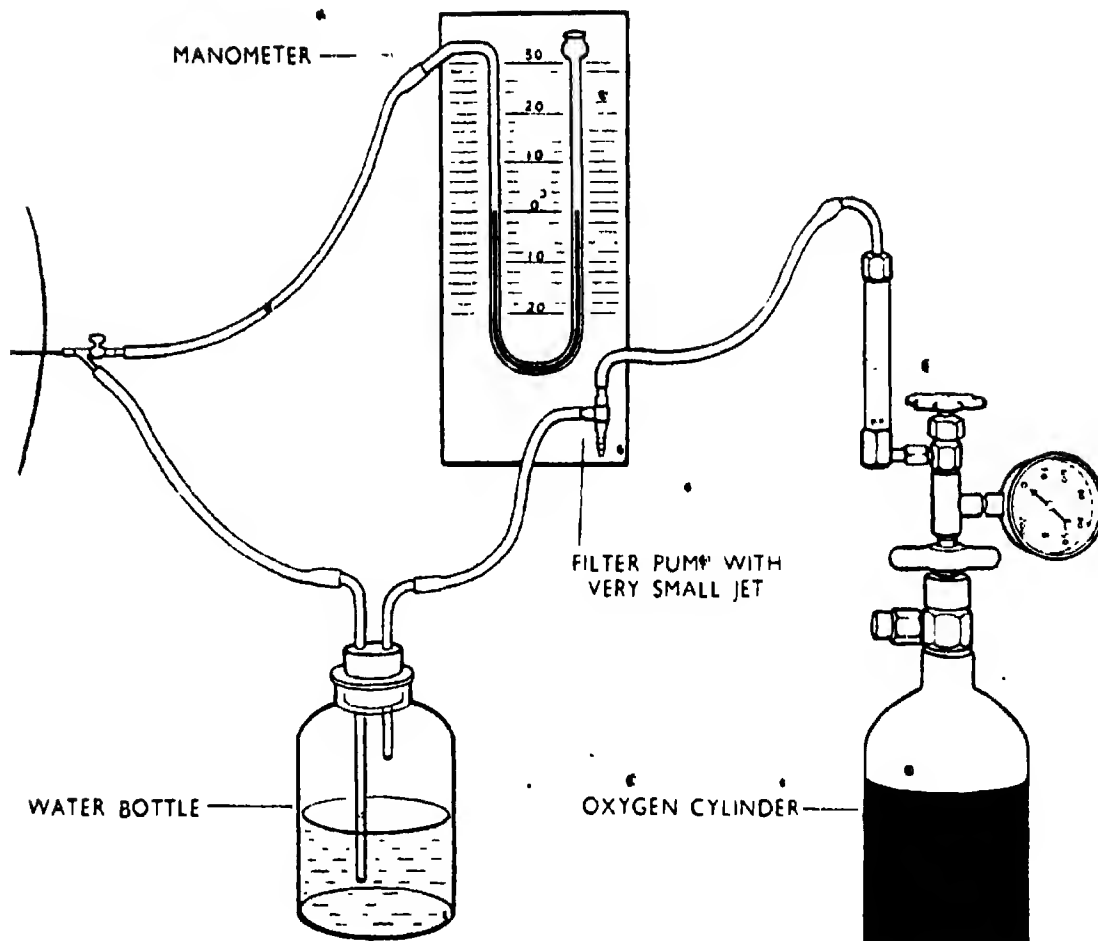


FIG 81

Diagram of apparatus for continuous suction of pneumothorax.

(3) Marriott and Foster-Carter's apparatus.

This apparatus consists of a large bottle, A (Fig. 82), capable of holding at least 80 fl. oz. (2.25 litres approx.) and having at the bottom an outlet, B, provided with a tap. This outlet tube must be straight and not curved downwards, otherwise it may act as a siphon. The neck of the bottle is closed with a rubber cork, pierced by a tube E, which dips below the level of the water in A and can slide in the cork, so that its height may be readily altered. When tap B is opened, water will flow from bottle A, and air will be drawn in through tube E. If E is closed, or if it communicates with a closed cavity, a negative pressure will develop within it, proportional to the height of its lower end X above the level Y of the outflow. Thus, if the distance XY is 10 cm., a negative pressure of 10 cm. of water will develop in E when tap B is opened.

When used in the treatment of spontaneous pneumothorax, the suction tube E is connected by rubber tubing with a pneumothorax needle. It is also convenient to incorporate a water manometer in the system to measure both the intra-pleural pressure when clip C is closed and the negative pressure developed by the apparatus when clip D is closed. Bottle A should be refilled with clip C closed, before the water falls below the level of X. A slight disadvantage of this apparatus is that, if the leak is large, the bottle has to be filled frequently.

(4) An electrical suction pump.

This should be capable of keeping up a low continuous suction.

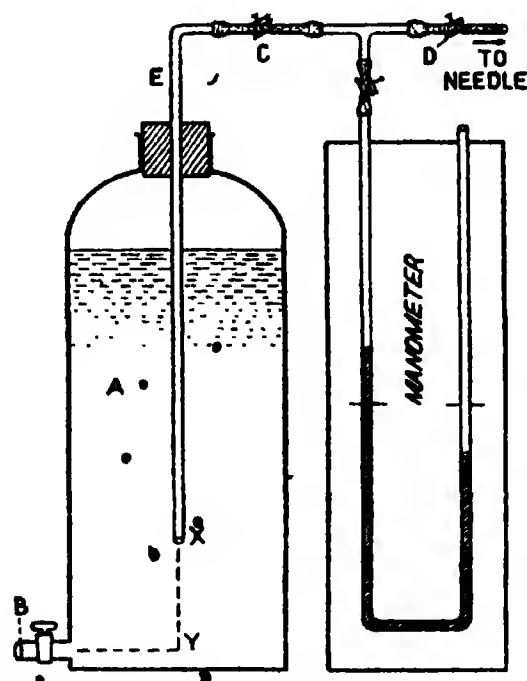


FIG. 82

Apparatus for continuous suction of pneumothorax.

TIDAL IRRIGATION OF THE PARALYSED BLADDER

The principle is that the bladder is slowly filled with a mild antiseptic solution from a container and automatically emptied at intervals by siphonage.

The irrigating fluid drips into the outer chamber (Figs. 83 and 84) and also passes to the bladder and into the ascending limb of the U-tube. When the fluid level in the outer chamber reaches the bend of the U-tube, the latter forms a siphon sucking out the contents of the bladder and of the outer chamber, which pass to the container. The height of the bend of the U-tube above the symphysis pubis determines the final pressure reached in the bladder. This should not be more than a few inches for completely paralysed bladders. As bladder tone improves, less fluid will be needed before siphonage begins and, when the bladder is able to empty itself reflexly, tidal irrigation can be discontinued. If, however, the residual urine is large in amount after reflex emptying, tidal irrigation should be restarted until reflex evacuation is more complete.

Note :

1. The rubber tube to the container should not lie under a considerable depth of fluid, as the increased pressure will be projected back to the patient's bladder.

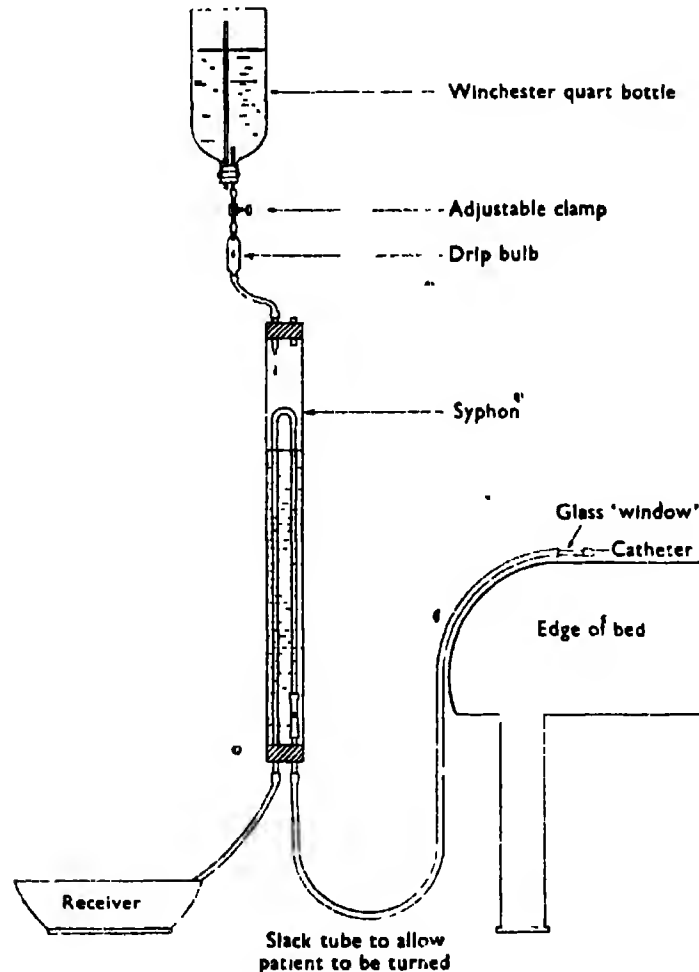


FIG 83

Apparatus for tidal irrigation of the bladder

2. Not less than 2 gallons (16 pints or 8 litres approximately) of irrigating fluid should pass through the apparatus in 24 hours, i.e., a Winchester "quart" bottle (4 pints or 2 litres approximately) should be used every six hours and an ordinary drip bulb will have to be *kept* running at two drops per second to deliver this amount.
3. In severe cystitis clots of pus may block the U-tube. In this case the outer tube will overflow through the air hole.

Supra-pubic route.

To avoid the disadvantages of the indwelling urethral catheter in the above method on the one hand and a supra-pubic cysto-

tomy on the other, tidal drainage may be applied through a small leakproof supra-pubic catheter. This requires a special technique for its introduction (Riches, E. W., *Lancet*, 1943, 1, 128).

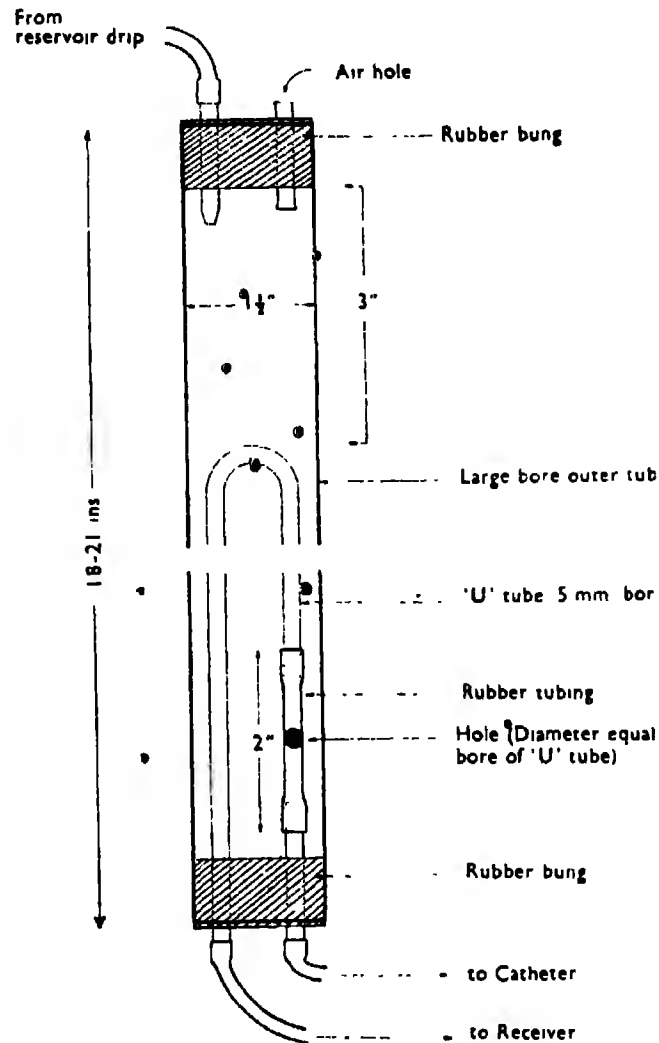


FIG. 84

Showing details of the syphon chamber.

GASTRIC LAVAGE

Position of patient.

In an unconscious patient with impending respiratory failure the insertion of a cuffed endo-tracheal tube by an anæsthetist is a useful preliminary to gastric lavage. Failing this the risk of fluid entering the lungs should be avoided by having the head low. A cuffed œsophageal tube is an additional safeguard. The patient

should be placed prone with his head over the table (Fig. 85), or in the Trendelenburg position on an operating table (Fig. 86). In the latter position, it is necessary to draw the tongue forwards



FIG. 85

Gastric lavage on an operating table. A. Stomach tube
B. Large safety pin in wall of tube. C. Gag. D. Sucker
tube. E. Tongue forceps F. Electric sucker

by a clip and to remove fluid from the mouth by a sucker. A struggling patient may have to be immobilised by straps, or by wrapping him tightly in a blanket.

Technique.

Dentures are removed and the mouth opened by a gag or box-wood wedge with a central hole (Fig. 87). A fairly stiff oesophageal



FIG 86

Gastric lavage, showing the proper position of the patient. The head must be lower than the rest of the patient. A. Stomach tube. B. Safety pin in wall of tube. C. Gag.

tube (not a Ryle's tube), preferably 60 inches long and about half an inch in diameter (for an adult) is lubricated, several large holes having been cut in its wall near the end. It is then passed

through the hole in the wedge if this is used, over the tongue and quickly down the œsophagus. The end of the tube should be 20 inches from the incisor teeth in an adult, and 10 inches in an



FIG. 87

Boxwood wedge with central hole to take œsophageal tube.



FIG. 88

" Puretha " respirator.
(Siebe, Gorman and Co. Ltd.)

infant. It is an advantage to mark these distances on the tube by a safety pin in its wall (but not in its lumen). A Ryle's tube should only be used if it is impossible to open the mouth. It may then be passed through the nose.

When the tube enters the stomach it is useful first to attach a Senoran's evacuator (Fig. 89) to empty the stomach. Then a funnel is attached and warm water is poured in. Bicarbonate solution may be used but not in barbiturate poisoning (*see page*

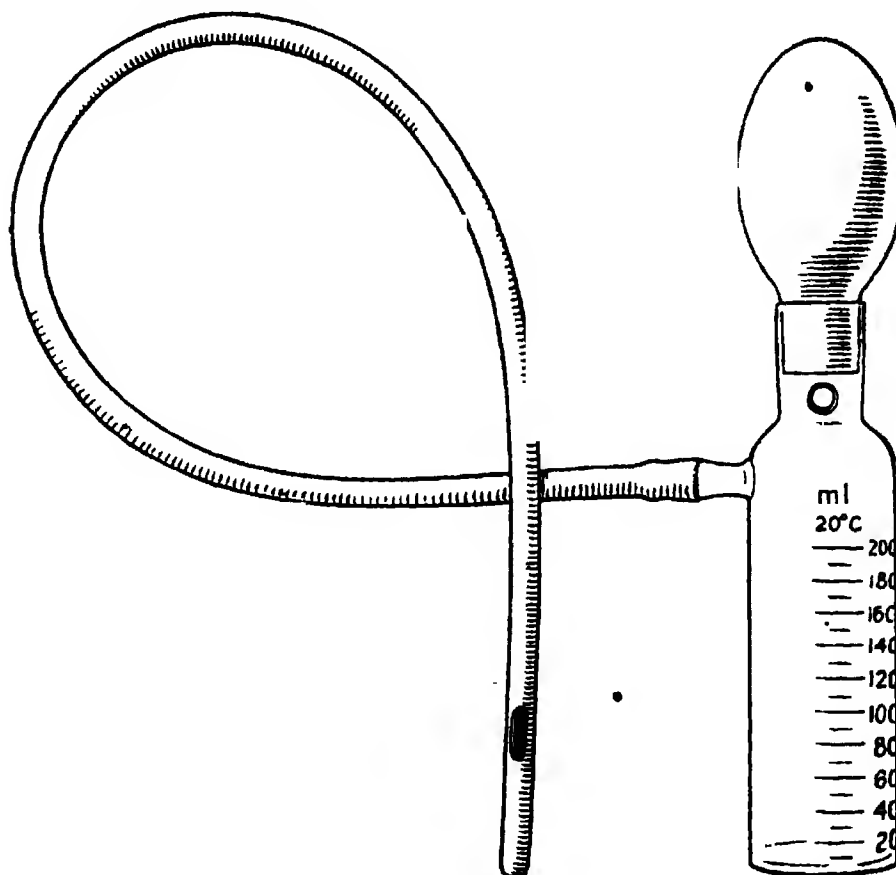


FIG 89
Senoran's evacuator.

11). After about half a pint has entered (less in children) but while the level of water is still visible in it, the funnel is lowered over a pail on the floor, and the fluid siphoned out of the stomach. Larger amounts may force the pylorus and so defeat the object of gastric lavage. A total amount of two gallons should be used, and the first washings should be preserved separately in case analysis is required.

RESPIRATORS

Three main types are available for rescue purposes. At least two models appropriate to the industry concerned should always be provided, and workmen should be practised in their use.

- (1) Canister types such as the "Puretha" (Fig. 88). These can be relied on for short periods in low concentrations of gas. Numerous canisters of distinctive colour are provided to give protection against different gases. Appropriate canisters should be available in any given industry.



FIG. 90

"Antipoy's" short distance breathing apparatus.
(Siebe, Gorman and Co. Ltd.)

- (2) Fresh air apparatus. "The Antipoy's" (Fig. 90). This has the same facepiece as the "Puretha" respirator and is connected to an air pipe 30 feet (9 metres approx.) long, the end of which must be in fresh air.
- (3) Self-contained oxygen apparatus. "The Salvus" (Fig. 91). This apparatus enables the wearer to remain up to half an hour in an atmosphere of 100 per cent. irrespirable air. It delivers oxygen automatically at 2 litres per minute and there

is a relief valve to deflate the breathing bag should this become over-inflated. Eye protecting goggles should be worn also in smoke or gases affecting the eyes.



FIG. 91

"Salvus" self contained oxygen
breathing apparatus.

(Siebe, Gorman and Co. Ltd.)

ARTIFICIAL RESPIRATION

Sudden failure of respiration demands prompt treatment, for if breathing has stopped for ten minutes death is almost certain and may even occur after a two-minute stoppage. After rescue from drowning or electrocution artificial respiration must be started at once. *Delay is dangerous and there is literally not one second to lose.* Don't stop to remove dentures, loosen clothing or drain the lungs. All these can be attended to later or by an assistant.

No one method of artificial respiration is universally applicable and the choice will depend on which one the operator knows, whether he is single-handed or not, and how much space is available. "Push and pull" methods are twice as effective as simple "push" methods, since they cause active inspiration as well as expiration. Success depends on the time factor, and so the best method is whichever the operator can promptly and persistently apply. Don't hurry with artificial respiration. If you do you will not only exhaust yourself but you may delay the onset of normal respiration and return to consciousness by lowering the CO_2 tension of the patient's blood through hyperventilation.

The main methods are:—

- (1) Holger Nielsen's arm lift - back pressure method (a "push and pull" method for a single operator). This causes better ventilation than other methods and also avoids the danger of pressure on the stomach causing regurgitation and inhalation of water. It is now recommended by the British Red Cross Society.
- (2) Schäfer's prone-pressure method (a "push" method for a single operator). This might have to be used if injuries to the arms and ribs prevented the use of the Holger Nielsen method. It is only for this reason that it is described here.
- (3) Drinker's arm-lift—a prone-pressure method. (Eve's modification—a "push and pull" method for two operators).
- (4) Mouth-to-mouth insufflation.
- (5) Eve's rocking method.

Holger Nielsen's method.

The chest is compressed against the ground for expiration and raised by the arms for inspiration.

1. Turn the patient face downwards with arms upwards and elbows flexed so that his head is turned to the side and rests on his hands.
2. Kneel on one knee at the patient's head and facing his feet. The opposite foot is near the patient's elbow (Fig. 92).
3. Inspiration. Grasp the arms above the elbows and rock backwards (Fig. 93) raising the arms until tension is felt. Count 1, 2, 3 while doing this ($= 2\frac{1}{2}$ seconds).

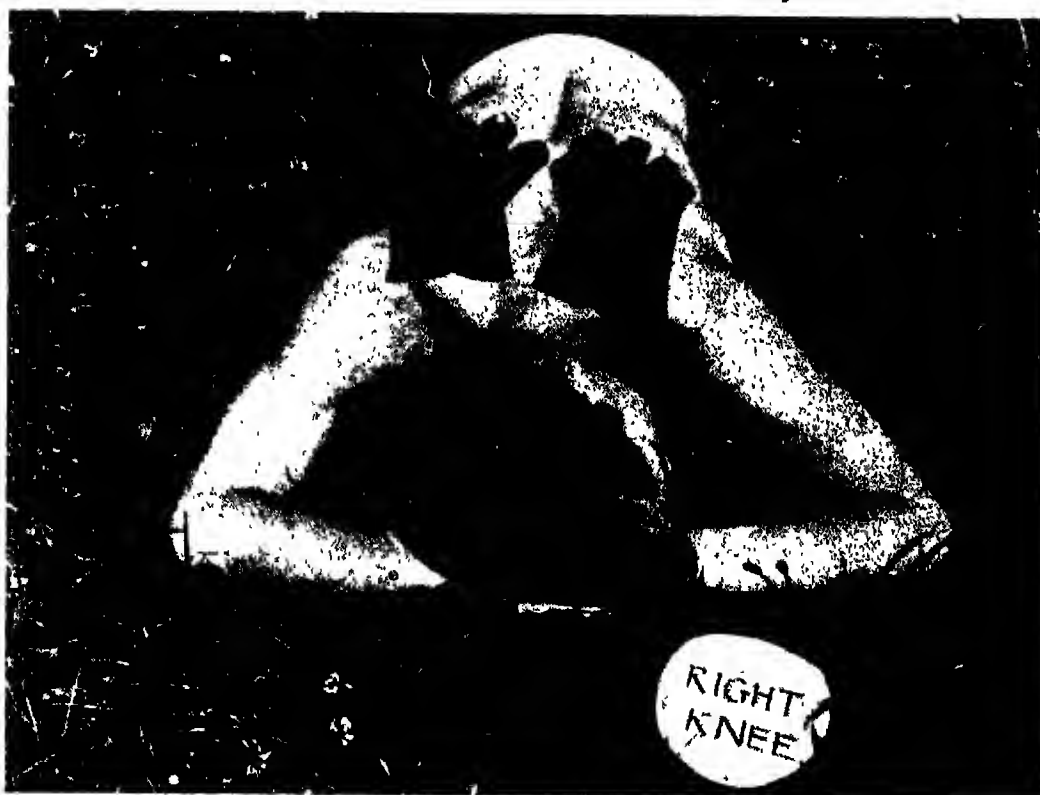


FIG. 92

Holger Nielsen method. Showing the position of the operator's hands (shown as gloves); his left foot (shown as a shoe); his right knee and also the correct position of the patient's arms and head.



FIG. 93

Holger Nielsen method. Inspiration. Duration $2\frac{1}{2}$ secs.

4. Expiration. Drop the arms (Fig. 94) and put your hands on the patient's back just below the scapulæ with your thumbs touching (Fig. 95) counting 4, as you do it. Rock forwards



FIG. 94

Holger Nielsen method Patient's arms dropped at end of inspiration.

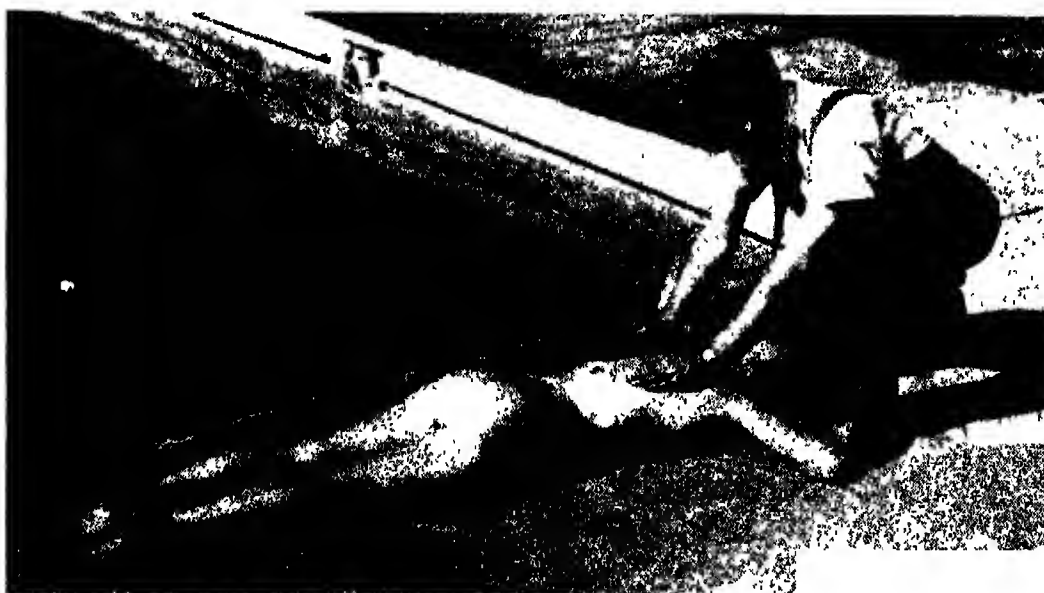


FIG. 95

Holger Nielsen method. Position of operator's hands and arms at beginning of expiration.

with your elbows straight and exert steady pressure on the chest (Fig. 96). Count 5, 6, 7 while doing this ($= 2\frac{1}{2}$ seconds).

5. Slide your hands off the back and on to the patient's arms as you count 8.

This cycle should be repeated 10 to 12 times a minute.

If the arm is injured inspiration should be achieved by lifting from the shoulders rather than from the upper arms.



FIG. 96

Holger Nielsen method. Expiration. Operator rocks forward with his arms straight. Duration $2\frac{1}{2}$ secs.

Schäfer's method.

1. Turn the patient face downwards with one arm extended overhead and the other bent at the elbow. His face is turned outwards and rests on his hand.
2. Kneel, straddling the patient's thighs or one thigh if this is too difficult, and place the palms of your hands on the small of his back with the tips of your fingers just out of sight.
3. Expiration. Keeping your arms straight swing forwards slowly so that the weight of your body is gradually brought to bear on the patient. This takes two seconds. (Count "one thousand, two thousand" or "one chimpanzee" and so on). Your shoulder should be directly over your wrist at the end of the forward swing.
4. Inspiration. Swing backwards for three seconds to remove the pressure completely. After one or two seconds swing forwards again.

The important point in Schäfer's method is that expiration depends on compression of the abdominal viscera pushing the diaphragm upwards. The operator should feel that the loins are yielding. His hands should therefore be placed low down over the small of the back and not over the rigid part of the thorax. They should not be taken off the patient during the whole time of the procedure. Inspiration is effected by the elastic recoil of the thorax and diaphragm. The disadvantages of Schäfer's method are that the operator cannot see what is happening so that it may degenerate into a ritual and also if the patient is deeply unconscious and cold the diaphragm will be toneless and unable to cause inspiration by elastic recoil. Inspiration should therefore be assisted by a second operator who lifts the arms (Drinker's method. See below). If there is a third assistant he should determine by feeling and listening that air is going in and out, and should also help by rubbing the limbs towards the heart. Relays of operators are necessary as the method is tiring.



FIG. 97

Drinker's arm-lift—prone-pressure method. Eve's modification. Expiration is produced by one operator using Schäfer's method



FIG. 98

Drinker's arm-lift—prone-pressure method. Eve's modification. Inspiration.

One operator using Schäfer's method takes pressure off the loins while the second operator assists inspiration by pulling up the patient's arms until the upper abdominal wall is off the ground. The patient's head is supported on the operator's knees,

Drinker's arm-lift—prone pressure method. (Eve's modification).

Professor Drinker suggested that inspiration (the weak point of Schäfer's method) could be improved by having a second operator to pull the arms towards the head as in the Danish method of Nielsen. Eve still further improved the method by having the arms *lifted* upwards (*i.e.* off the ground). The technique is as follows:—

One operator applies Schäfer's method for expiration (Fig. 97). The other kneels at the patient's head and assists inspiration by lifting the arms upwards (Fig. 98) so that the abdominal wall is just clear of the ground. This allows the upper abdominal contents to sag and causes the diaphragm to be pulled down.

Mouth-to-mouth method.

This ancient method mentioned in the Bible (II Kings IV: 34) consists in blowing up the lungs and while it provides adequate tidal air it is generally avoided as being æsthetically disagreeable. It is, however, very useful in children for asphyxia other than from drowning and in the newly born.

Compress the patient's nostrils with your right hand. Support his jaw with the fourth and fifth fingers of your left hand and with your thumb and other two fingers of the left hand cup his mouth. Considerable pressure between your lips and his is required. An airway is an advantage as the tongue may fall back.

Eve's rocking method.

Eve has pointed out that manual methods of artificial respiration depend on the elastic recoil of the thoracic wall and diaphragm for inspiration, and that this may be absent in the drowned because of loss of muscular tone. In such cases a rocking method is advisable. Besides causing efficient ventilation, it has the advantage of aiding circulation. Wet clothes can be replaced and dressings applied while rocking goes on. Relays of trained operators are not needed.

Rocking may be achieved in various ways:—

1. On specially designed stretchers or beds (Fig. 99).
2. On improvised rocking stretchers.

A door or ladder seven feet long is obtained and the patient lashed to it. For a trestle a low fence, or the backs of two chairs, or a loop of rope may be used—all of which should

Appendix I

Vaccines, Sera and Anti-Venines

Sera for emergency treatment are now available through certain hospitals, and for prophylaxis, where there is less urgency, through certain laboratories. (The addresses and telephone numbers have been corrected to August 31, 1956).

TREATMENT OF ANTHRAX, BOTULISM and SNAKE BITE

Revised list of centres which keep emergency supplies of sera for the treatment of anthrax, botulism and snake-bite. A 24-hour service is maintained at each centre.

Region 1: Newcastle

The Dispensary, Cumberland Infirmary, Carlisle .	Carlisle 22332
The Dispensary, Newcastle General Hospital .	Newcastle 35211

Region 2: Leeds

Castle Hill Hospital, Cottingham, Hull . . .	Cottingham 47372
Seacroft Hospital, York Road, Leeds . . .	Leeds 45194

Region 3: Sheffield

Nottingham City Hospital, Hucknall Road, Nottingham	Nottingham 63361
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Region 4: East Anglia

Regional Blood Supply Depot, Brookland Avenue, Cambridge	
Monday—Friday 8.30 a.m.—5.30 p.m. }	Cambridge 56912
Saturday 8.30 a.m.—12.30 p.m. }	
All other times	

Region 6: N.E. Metropolitan

North Middlesex Hospital, N.18	
Day—Chief Pathologist }	Edmonton 3071
Night—Resident Pathologist }	

Region 7: S.E. Metropolitan

South East Sub-Depot, Weaving House, Ashford Road, Maidstone, Kent	
Day Service	{ Maidstone 4468
	{ Maidstone 3604
Night Service	{ Maidstone 4660
	{ Maidstone 3718

Region 8: S.W. Metropolitan

South London Blood Supply Depot, Stanley Road, Sutton, Surrey	Vigilant 8221
The Dispensary, Royal South Hants and Southampton Hospital, (†Snake Anti-Serum only) .	Southampton 26211

Region 9: Oxford

Northampton General Hospital	Northampton 4680
Royal Berkshire Hospital, Reading	Reading 81721

Region 10: South Western

Ham Green Hospital, Bristol	Bristol 31165
Pathological Department, Royal Devon and Exeter Hospital, Exeter	{ Exeter 3519
Scott Isolation Hospital, Plymouth	{ Exeter 2261*
Pathological Department, Royal Cornwall Infirmary, Truro	Plymouth 64311
	Truro 3029

Region 11: Welsh

Cærnarvon and Anglesey General Hospital, Bangor (Botulinum and Snake-bite Antisera only)	Bangor 1181
City Isolation Hospital, Cardiff	Cardiff 21466
West Wales General Hospital Glanwili, Carmarthen (Botulinum and Snake-bite Antisera only)	Carmarthen 7433

Region 12: Birmingham

The Dispensary, Selly Oak Hospital, Birmingham	Selly Oak 1361
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Region 13: Manchester

The Dispensary, Manchester Royal Infirmary	Ardwick 3300
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Region 14: Liverpool

The Fazakerley Isolation Hospital, Liverpool	Aintree 2324
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* When laboratory closed.

† This Hospital carries anti-snake serum, not only against native snakes but against tropical snakes which may be imported with cargoes arriving at the Docks.

Anthrax antiserum for *prophylactic* use may be obtained from:

Place	Address	Telephone Number
Bradford	Public Health Laboratory, 16-18 Edmund Street	Bradford 24314
Cardiff	Public Health Laboratory, Institute of Preventive Medicine, The Parade	Cardiff 29110
Hull	Public Health Laboratory, 184 High Street	Hull (Central) 35371
Liverpool	Public Health Laboratory, 126 Mount Pleasant	Royal 3636
London	Central Public Health Laboratory, Colindale Avenue, N.W.9	Colindale 7041
Newcastle	Public Health Laboratory, General Hospital, Westgate Road	Newcastle 34920
Northampton	Public Health Laboratory, General Hospital	Northampton 347
Taunton	Public Health Laboratory, Musgrove Park Hospital	Taunton 5753
Worcester	Public Health Laboratory, Royal Infirmary	Worcester 2697

"MUSHROOM" POISONING (see page 73).

At the time of writing no stocks of anti-phallic serum are held in Britain and it would have to be obtained in an emergency from Institut Pasteur, 28 Rue du Dr Roux, Paris XV^e (Tel. SEGur 01-10). Air transport can be arranged through (1) British European Airways, Dorland House, 14 Regent Street, S.W.1. (Tel. GERard 9833) or, at London Airport (Tel. HOUns-

low 7711), or (2) At France, 52 Haymarket, S.W.1. (Tel. WHItchall 4455) or, at London Airport, HOUnslow 7711 Extension 235. The French Embassy in London (58 Knightsbridge, S.W.1. Tel. SLOane 3404) will always help in arranging to send serum by the quickest route. In case of any difficulty in Paris the British Embassy should be approached (35 Rue du Faubourg Saint-Honore, Paris 8^e. Tel. ANJou 2710).

OTHER SUPPLIERS OF ANTI-VENINES FOR SNAKE BITE

Great Britain.

Allen and Hanburys Ltd., 7 Vere Street, London W.1. Tel. GROsvenor 7571. Hours 8.30 a.m. to 6 p.m. (1 p.m. Saturdays). Closed on Sundays and Bank Holidays.
(Anti-venine against the common adder, *Vipera berus*, the only poisonous snake in Britain).

United States of America.

The sole producers of anti-venines for snake-bite in U.S.A. are Wyeth Laboratories, 1401 Walnut Street, Philadelphia 2. Pa. U.S.A. Tel. Locust 4-2800.
(The serum is polyvalent and supplied as a dry powder with a syringe and sterile water for reconstitution).

India.

The Central Research Institute, Kasauli, P.E.P.S.U. India. Tel. Kasauli 62. Telegrams "Problem."
(Polyvalent anti-venine against the cobra and Russell's viper).
The Director, Haffkine Institute, Parel, Bombay, 12. Tel. 60084. Telegrams "Research."
(Lyophilised polyvalent serum against cobra, common krait, Russell's viper and the saw-scaled viper).

Australian Capital Territory.

Director-General of Health, Canberra, A.C.T. Tel. Canberra FO 611. Telegrams "Health, Canberra."

New South Wales.

Commonwealth Deputy Director of Health, Erskine House, 39 York Street, Sydney. Tel. BX 1251.
The Medical Officer-in-Charge, Health Laboratory, Lismore, N.S.W. Tel. Lismore 346.

Queensland.

Deputy Director of Health, Commonwealth Department of Health, Anzac Square, Adelaide Street, Brisbane. Tel. FA 0101 Ext. 423 (8.45 a.m. to 5.06 p.m.). After office hours Tel. MV 1271. Telegrams "Quarantine, Brisbane."
Medical Officer-in-Charge, Health Laboratory, P.O. Box 487, Townsville. Tel. 5108. (After office hours 4913).
Medical Officer-in-Charge, Health Laboratory, P.O. Box 36, Toowoomba. Tel. Toowoomba 914.

Medical Officer-in-Charge, Health Laboratory, Quay Lane, P.O. Box 301, Rockhampton. Tel. Rockhampton 4232. (After office hours 2267).

Medical Officer-in-Charge, Commonwealth Health Laboratory, P.O. Box 672, Cairns. Tel. 2467.

South Australia.

Deputy Director of Health, Commonwealth Department of Health, Box 1790 N, G.P.O., Adelaide, S. Australia. Tel. LA 2666.

Biochemist-in-Charge, Health Laboratory, Port Pirie, S. Australia. Tel. 353. (After office hours 368).

Western Australia.

Deputy Director of Health, 473 Wellington Street, Perth W.A. Tel. BA 8211.

Medical Officer-in-Charge, Commonwealth Health Laboratory, Maritana Street, Kalgoorlie. Tel. 173.

Victoria.

Medical Officer-in-Charge, Health Laboratory, Bendigo. Tel. Bendigo 867.

Commonwealth Serum Laboratories, Parkville, N.2., Victoria. Tel. F.W. 2101. Telegrams "Serums," Melbourne.

Tasmania.

Deputy Director, Commonwealth Department of Health, Commonwealth Offices, Stowell Avenue, Battery Point, Hobart, Tasmania. Tel. B 2661. (After office hours B 1838).

Medical Officer-in-Charge, Commonwealth Health Laboratory, Corner Howick and Mulgrave Street, Launceston, Tasmania. Tel. B 3589. (After office hours East 4470).

South Africa.

The South African Institute of Medical Research,

- (i) P.O. Box 1038, Johannesburg. Tel. 44-1444. Telegrams "Bacteria."
- (ii) Buckingham Road, Port Elizabeth, Cape. Tel. 6571 and 6505. Telegrams "Bacteria."
- (iii) 7 Roth Avenue, Bloemfontein, Orange Free State. Telegrams: "Bacteria."

The Institute also supplies serum to the Government Departments of Nigeria, Gold Coast, Belgian Congo, French Equatorial Africa, Portuguese East and West Africa, French Cameroons, and the numerous missionary stations in Central Africa.

(Polyvalent anti-venine against crotaline and lachesine vipers).

South America.

Laboratorios Butantos, 34 Rua Gloria, Sao Paulo, Brazil. Tel. Sao Paulo 2-1788.

(Polyvalent anti-venine against crotaline and lachesine vipers).

EMERGENCY PROPHYLAXIS

The following Constituent and Associated Laboratories of the Public Health Laboratory Service in England and Wales hold stocks of certain prophylactic materials not readily obtainable through trade sources.

A.—CONSTITUENT LABORATORIES ENGLAND

<i>Place</i>	<i>Address</i>	<i>Pathologist in charge</i>	<i>Telephone number</i>
Bath ...	Public Health Laboratory, Manor Hospital, Combe Park, Bath.	Dr. P. G. Mann	Bath 7250
Bedford ...	Public Health Laboratory, General Hospital, Kim- bolton Road, Bedford.	Dr. W. F. Lane	Bedford 66896
Birkenhead ...	Public Health Laboratory, 42 Hamilton Square, Birkenhead.	Dr. J. M. Ritchie	Birkenhead 5484
Birmingham..	Public Health Laboratory, 150 Great Charles Street, Birmingham 3.	Dr. B. R. Sandiford	Central 6921/2
Bournemouth	Public Health Laboratory, Gloucester Road, Bos- combe, Bournemouth.	Dr. G. J. G. King	Boscombe 37304
Bradford ..	Public Health Laboratory, 16-18 Edmund Street, Bradford.	Dr. H. G. M. Smith	Bradford 24314
Brighton .	Public Health Laboratory, Royal Sussex County Hospital, Brighton 7.	Dr. J. E. Jameson	Brighton 3506
Cambridge ...	Public Health Laboratory, Tennis Court Road, Cam- bridge.	Dr. R. M. Fry	Cambridge 55526
Carlisle ...	Public Health Laboratory, Cumberland Infirmary, Carlisle.	Dr. D. G. Davies	Carlisle 22332
Coventry ...	Public Health Laboratory, Stoney Stanton Road, Coventry.	Dr. R. E. Jones	Coventry 63256-7 and 5937
Dorchester ...	Public Health Laboratory, Glyde Path Road, Dor- chester, Dorset.	Dr. G. H. Tee	Dorchester 1278
Epsom ...	Public Health Laboratory, West Hill House, West Hill, Epsom, Surrey.	Dr. D. M. Stone	Epsom 2474
Exeter ...	Public Health Laboratory, 7 Dix's Field, Exeter.	Dr. B. Moore	Exeter 54959
Guildford ...	Public Health Laboratory, St. Luke's Hospital, Guild- ford.	Dr. G. T. Cook	Guildford 66091
Harrogate ...	Public Health Laboratory, Harrogate and District General Hospital.	Dr. D. J. H. Payne (part- time)	Harrogate 84077
Hereford ...	Public Health Laboratory, County Hospital, Hereford.	Dr. D. R. Christie	Hereford 4696
Hull .	Public Health Laboratory, 184 High St., Kingston- upon-Hull.	Dr. J. McCoy	Hull Central 35371
Ipswich ...	Public Health Laboratory, Borough General Hospital, Woodbridge Road, Ipswich.	Dr. P. H. Martin	Ipswich 77261/2

<i>Place</i>	<i>Address</i>	<i>Pathologist in charge</i>	<i>Telephone number</i>
Leeds ...	Public Health Laboratory, Bridle Path, York Road, Leeds 15.	Dr. G. B. Ludlam	Leeds 645011
Leicester ...	Public Health Laboratory, Isolation Hospital, Groby Road, Leicester.	Dr. N. S. Mair	Anstey 2383
Lincoln ...	Public Health Laboratory, St. Anne's Road, Lincoln.	Dr. J. M. Croll	Lincoln 8607
Liverpool ...	Public Health Laboratory, 126 Mount Pleasant, Liver- pool 3.	Prof. D. T. Robinson	Royal 3636
London (Colindale)	Central Public Health Laboratory, Colindale Avenue, London N.W.9.	Lt.-Col. H. J. Bensted	Colindale 7041
London (County Hall)	Bacteriological Laboratory, (M.R.C.), Room 617, County Hall, Westminster Bridge, S.E.1.	Dr. A. J. H. Tomlinson	Waterloo 3467
Luton ...	Public Health Laboratory, Luton and Dunstable Hos- pital, Lewsey Road, Luton.	Dr. H. D. Holt	Luton 6807
Maidstone ...	Public Health Laboratory, County Hall, Maidstone.	Dr. J. H. C. Walker	Maidstone 4321
Manchester ...	Public Health Laboratory, Monsall Hospital, Newton Heath, Manchester 10.	Dr. M. T. Parke	Collyhurst 2733
Middles- brough ...	Public Health Laboratory, General Hospital, Ayre- some Green Lane, Middles- brough.	Dr. A. R. Blowers	Middles- brough 87766
Newcastle-on- Tyne	Public Health Laboratory, General Hospital, West- gate Road, Newcastle-on- Tyne 4.	Dr. A. I. Messer	Newcastle 34920
Northallerton..	Public Health Laboratory, Friarage Hospital, North- allerton, Yorks.	Dr. D. J. H. Payne	Northall- erton 88
Northampton..	Public Health Laboratory, General Hospital, North- ampton.	Dr. L. Hoyle	Northamp- ton 347
Norwich ...	Public Health Laboratory, Bowthorpe Road, Norwich.	Dr. L. M. Dowsett	Norwich 28145
Nottingham...	Public Health Laboratory, 63 Goldsmith Street, Nottingham.	Dr. E. R. Mitchell	Nottingham 46436
Oxford ...	Public Health Laboratory, Walton Street, Oxford.	Dr. R. L. Vollum	Oxford 47884/5
Peterborough	Public Health Laboratory, Peterborough and District Memorial Hospital, Mid- land Road, Peterborough.	Dr. C. C. B. Gilmour	Peter- borough 2277
Plymouth ...	Public Health Laboratory, South Devon and East Cornwall Hospital, Green- bank Road, Plymouth.	Dr. C. H. Jellard	Plymouth 63997

<i>Place</i>	<i>Address</i>	<i>Pathologist in charge</i>	<i>Telephone number</i>
Portsmouth ...	Public Health Laboratory, Central Laboratory, Milton Road, Portsmouth.	Dr. K. E. Hughes	Portsmouth 74785/6/7
Reading ...	Public Health Laboratory, Battle Hospital, Reading.	Dr. N. Wood	Reading 2957
Salisbury ...	Public Health Laboratory, General Infirmary, Salis- bury.	Dr. M. S. Pereira	Salisbury 2950
Sheffield ...	Public Health Laboratory, City General Hospital, Sheffield 5.	Dr. E. H. Gillespie	Sheffield 36253
Shrewsbury ...	Public Health Laboratory, Royal Salop Infirmary, Shrewsbury.	Dr. A. C. Jones	Shrewsbury 4389
Southampton..	Public Health Laboratory, The Health Centre, King's Park Road, Southampton.	Dr. R. I. Hutchinson	Southamp- ton 3788
Southend ...	Public Health Laboratory, Westcliff Hospital, Bal- moral Road, Southend-on- Sea.	Dr. R. Pilsworth	Southend 45440
Stafford ...	Public Health Laboratory, Martin Street, Stafford.	Dr. R. N. Phease	Stafford 377
Sunderland ...	Public Health Laboratory, Havelock Hospital, Hylton Road, Sunderland.	Dr. P. B. Cline	Sunderland 4462
Taunton ...	Public Health Laboratory, Musgrove Park Hospital, Taunton.	Dr. J. A. Boycott	Taunton 5753
Truro ...	Public Health Laboratory, Royal Cornwall Infirmary, Truro.	Dr. F. D. M. Hocking	Truro 3029
Wakefield ...	Public Health Laboratory, County Medical Offices, Wood Street, Wakefield, Yorks.	Dr. L. A. Little	Wakefield 2207
Watford ...	Public Health Laboratory, Peace Memorial Hospital, Watford.	Dr. B. H. E. Cadness-Graves	Watford 2369
Winchester ...	Public Health Laboratory, Royal Hants County Hospital, Winchester.	Brig. H. T. Findlay	Winchester 3807
Worcester ...	Public Health Laboratory, Royal Infirmary, Worces- ter.	Dr. R. J. Henderson	Worcester 5238/9

WALES

<i>Place</i>	<i>Address</i>	<i>Pathologist in charge</i>	<i>Telephone number</i>
Aberystwyth...	Public Health Laboratory, Cardiganshire General Hospital, Aberystwyth.	Dr. M. V. N. Sidds	Aberyst- wyth 216
Cardiff ...	Public Health Laboratory, Institute of Preventive Medicine, The Parade, Cardiff.	Prof. Scott Thomson	Cardiff 29110 and 23967
Carmarthen ...	Public Health Laboratory, Penlan Road, Carmarthen.	Dr. W. Kwantes	Carmarthen (7271
Conway ...	Public Health Laboratory, Bryn Hyfryd, Conway.	Dr. A. J. Kingsley Smith	Conway 2178
Newport (Mon.) ...	Public Health Laboratory, County Hall, Newport, Mon.	Dr. R. D. Gray	Newport 65431

B.—ASSOCIATED LABORATORIES

ENGLAND

Bristol ...	Department of Preventive Medicine, Whatley Road, Clifton, Bristol 8.	Prof. K. E. Cooper	Bristol 38257
Derby ...	County Bacteriological Lab- oratory, County Offices, St. Mary's Gate, Derby.	Dr. J. L. G. Iredale	Derby 47131 Ext. 120
*Leeds ...	Bacteriological Department, School of Medicine, Leeds 2.	Prof. C. L. Oakley	Leeds 20071 Ext. 11

WALES

*Swansea	Pathological Department, Beck Laboratory, General Hospital, Swansea.	Lt.-Col. H. C. M. Walton	Swansea 2001
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* To be replaced by a constituent laboratory.

C.—REFERENCE LABORATORIES

Central Enteric Reference Laboratory and Bureau:

Director: Dr. E. S. Anderson.*Address:* Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 7041.

Dysentery Reference Laboratory:

Acting Director: Dr. K. Patricia Carpenter.*Address:* Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 7041.

Malaria Reference Laboratory:

Director: Sir Gordon Covell.*Address:* Horton Hospital, Epsom. *Tel.:* Epsom 2343.

Mycological Reference Laboratory:

Acting Director: Dr. Jacqueline Walker.*Address:* London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C.1. *Tel.:* Museum 3041.

Salmonella Reference Laboratory:

Director: Dr. Joan Taylor.*Address:* Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 7041.

Streptococcus and Staphylococcus Reference Laboratories:

Director: Dr. R. E. O. Williams.*Address:* Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 7041.

Venereal Diseases Reference Laboratory:

Director: Dr. I. N. Orpwood Price.*Address:* "X" Block, Eastern Hospital, Homerton Grove, E.9. *Tel.:* AMHerst 1532.

Virus Reference Laboratory:

Director: Dr. F. O. MacCallum.*Address:* Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 7041.

D.—SPECIAL LABORATORIES

Air Hygiene Laboratory:

Director: Dr. R. E. O. Williams.*Address:* Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 7041.

Epidemiological Research Laboratory:

Director: Dr. W. Charles Cockburn.*Address:* Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 7041.

Food Hygiene Laboratory:

Director: Dr. Betty C. Hobbs.*Address:* Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 7041.

National Collection of Type Cultures:

Director: Dr. S. T. Cowan.*Address:* Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 7041.

Standards Laboratory for Serological Reagents:

Director: Lt.-Col. H. J. Bensted.*Address:* Central Public Health Laboratory, Colindale Avenue, London, N.W.9. *Tel.:* Colindale 7041.

E. --REFERENCE EXPERTS

(For the examination of materials other than those normally submitted to the Reference laboratories listed above).

- Dr. C. H. Andrewes (Influenza):
National Institute for Medical Research, The Ridgeway, Mill Hill,
London, N.W.7. Tel.: Mill Hill 3866.
- Lt.-Col. H. J. Bensted (Meningococci):
Central Public Health Laboratory, Colindale Avenue, London, N.W.9.
Tel.: Colindale 7041.
- Dr. J. C. Broom (Leptospiral infections):
Wellcome Laboratories for Tropical Medicine, 183 Euston Road,
London, N.W.1. Tel.: Euston 4688.
- Prof. J. J. C. Buckley (Helminthology):
London School of Hygiene and Tropical Medicine, Keppel Street,
London, W.C.1. Tel.: Museum 3041.
- Prof. P. A. Buxton (Entomology):
London School of Hygiene and Tropical Medicine, Keppel Street,
London, W.C.1. Tel.: Museum 3041.
- Dr. I. A. B. Cathie (Toxoplasmosis):
Department of Clinical Pathology, Hospital for Sick Children, Great
Ormond Street, London, W.C.1. Tel.: Holborn 9200, Ext. 24.
- Prof. J. C. Cruickshank (Anaerobes and Brucella):
London School of Hygiene and Tropical Medicine, Keppel Street,
London, W.C.1. Tel.: Museum 3041.
- Prof. A. W. Downie (Smallpox):
Bacteriology Department, School of Hygiene, Mount Pleasant, Liver-
pool, 3. Tel.: Royal 6022.
- Dr. J. A. Dudgeon (Toxoplasmosis):
Department of Clinical Pathology, Hospital for Sick Children, Great
Ormond Street, London, W.C.1. Tel.: Holborn 9200, Ext. 24.
- Lt.-Col. H. J. Bensted (Typhus fever):
Central Enteric Reference Laboratory, Central Public Health Labora-
tory, Colindale Avenue, London, N.W.9. Tel.: Colindale 7041.
- Dr. R. M. Fry (Typing of Group A streptococci):
Public Health Laboratory, Tennis Court Road, Cambridge. Tel.: Cam-
bridge 55526.
- Prof. P. C. C. Garnham (Protozoology):
London School of Hygiene and Tropical Medicine, Keppel Street,
London, W.C.1. Tel.: Museum 3041.
- Dr. C. C. B. Gilmour (Cholera vibrios):
Public Health Laboratory, Peterborough and District Memorial
Hospital, Midland Road, Peterborough. Tel.: Peterborough 2277.
- Dr. Betty C. Hobbs (Food poisoning):
Food Hygiene Laboratory, Central Public Health Laboratory, Coli-
ndale Avenue, London, N.W.9. Tel.: Colindale 7041.
- Dr. L. Hoyle (Influenza):
Public Health Laboratory, General Hospital, Northampton. Tel.:
Northampton 347.
- Dr. M. T. Parker (Plague):
Public Health Laboratory, Monsall Hospital, Manchester, 10. Tel.:
Collyhurst 2733.
- Prof. D. T. Robinson (Diphtheria):
Bacteriological Department, City Laboratories, 126 Mount Pleasant,
Liverpool, 3. Tel. Royal 3636/7.

- Dr. Joan Taylor (Typing of strains of *Bact. coli* associated with infantile enteritis):
Salmonella Reference Laboratory, Central Public Health Laboratory,
Colindale Avenue, London, N.W.9. Tel.: Colindale 7041.
- Dr. Scott Thomson (Typing of Group A streptococci):
Public Health Laboratory, The Parade, Cardiff. Tel.: Cardiff 29110.
- Dr. A. J. H. Tomlinson (Anthrax):
Bacteriological Laboratory (M.R.C.), Room 617, County Hall,
Wes'minster Bridge, London, S.E.1. Tel.: Waterloo 3467.
- Dr. R. L. Vollum (Typing of Group A streptococci):
Public Health Laboratory, Walton Street, Oxford. Tel.: Oxford
47884/5.
- Dr. A. Q. Wells (Tubercle bacilli):
Sir William Dunn School of Pathology, South Parks Road, Oxford.
Tel.: Oxford 47626.
- Dr. R. E. O. Williams (Pneumococci):
Central Public Health Laboratory, Colindale Avenue, London, N.W.9
Tel.: Colindale 7041.

LIST OF SPECIALIST INVESTIGATIONS AVAILABLE TO ALL LABORATORIES OPERATING UNDER THE NATIONAL HEALTH SERVICE ACT, AND TO UNIVER- SITY LABORATORIES

Note 1.—The letter (A), (B), (C), etc., after an expert's name is a reference to the list, given above, in which his address will be found.

- 2.—In the following list the name of the expert who is responsible for the relevant examination is given; it should be added, however, that the serological diagnosis of *influenza*, *lymphogranuloma venereum*, *psittacosis* and *Q fever* is being undertaken regionally by the laboratories at Cambridge, Exeter, Hull, Manchester, Newcastle, Northampton, Nottingham, Winchester and Cardiff (see list A). Similarly, the serological diagnosis of *leptospiral infections* is being undertaken by the laboratories at Birmingham, Cambridge, Exeter, London (Colindale), Newcastle, Portsmouth and Cardiff.

<i>Anaerobes</i> , identification	Prof. J. C. Cruickshank (E).
<i>Anthrax bacilli</i> , identification	Dr. A. J. H. Tomlinson (E).
<i>Brucella</i> infections:		
(a) cultural tests	} Prof. J. C. Cruickshank (E).
(b) serological tests	
<i>Cholera vibrios</i> , identification	Dr. C. C. B. Gilmour (E).
<i>Diphtheria bacilli</i> , serological typing	Prof. D. T. Robinson (E).
<i>Dysentery bacilli</i> , typing	Dr. K. Patricia Carpenter (C).
<i>Encephalitis</i> , acute infectious	Dr. F. O. MacCallum (C).

Enteric Fever:

- (a) Vi - agglutination in suspected carriers
- (b) Vi-phage-type determination of strains of typhoid and paratyphoid bacilli Dr. E. S. Anderson (C).
- (c) Vi-phage-type determination of strains of *Salmonella typhimurium* (from outbreaks only)

Enteritis, infantile, typing of strains of

- Bact. coli* Dr. Joan Taylor (E).
- Entomological specimens, investigation* ... Prof. P. A. Buxton (E).
- Food Poisoning* Dr. Betty C. Hobbs (D).

Note: Owing to the perishable nature of most foodstuffs, material for investigation from outbreaks of food poisoning should normally be sent to the nearest public health laboratory. The reference laboratory should be used mainly for non-perishable articles of food, especially when litigation may arise, and for the identification of strains.

- Fungi (pathogenic), identification* ... Dr. Jacqueline Walker (C).
- Helminthological specimens, investigation* ... Prof. J. J. C. Buckley (E).

** Influenza:*

- (a) Cultural tests { Dr. C. H. Andrews (E).
- (From the area within a 30-mile radius of Northampton only) ... Dr. F. O. MacCallum (C).
- (b) serological tests:
 - (i) country north of a line joining the Wash and the mouth of the River Severn Dr. L. Hoyle (E).
 - (ii) country south of this line ... Dr. F. O. MacCallum (C).
- * Leptospiral infections, agglutination tests* ... Dr. J. C. Broom (E).
- Lymphocytic meningitis* Dr. F. O. MacCallum (C).
- * Lymphogranuloma venereum* Dr. F. O. MacCallum (C).
- Malaria* Sir Gordon Covell (C).
- Meningococci, typing* Lt.-Col. H. J. Bensted (E).
- Plague, investigation* Dr. M. T. Parker (E).
- Pneumococci, typing of from epidemics* ... Dr. R. E. O. Williams (E).
- Pneumonia, atypical* Dr. F. O. MacCallum (C).
- Poliomyelitis* Dr. F. O. MacCallum (C).
- Protozoological specimens, investigation* ... Prof. P. C. C. Garnham (E).
- * Psittacosis, diagnosis* Dr. F. O. MacCallum (C).
- * Q Fever* Dr. F. O. MacCallum (C).
- Salmonella organisms, typing* Dr. Joan Taylor (C).
- Smallpox, laboratory tests for diagnosis* ... Prof. A. W. Downie (E).
- Staphylococci, bacteriophage typing* ... Dr. F. O. MacCallum (C).
- Streptococci of Group A, typing:*
 - Streptococcus Reference Laboratory* ... Dr. R. E. O. Williams (C).
 - Regional typing laboratories:*
 - (i) *North:* Northumberland, Cumberland, Westmorland, Co. Durham, Lancs, Yorks Dr. R. E. O. Williams (C).
 - South East:* London, Middlesex, Kent, Surrey, Sussex, Hants, Dorset
 - (ii) *East:* Essex, Herts, Beds, Hunts, Cambs., Norfolk, Suffolk, Northants, Rutland, Leics., Derby, Notts, Lincs ... Dr. R. M. Fry (E).

* See Note 2 above.

(iii) <i>West:</i> Berks, •Bucks, Oxon, Warwick, Staffs, Cheshire, Salop, Worcs, Hereford, Glos, Wilts, Somerset, •Devon, Cornwall	Dr. R. L. Vollum (E).
• (iv) <i>Wales</i>	Dr. Scott Thomson (E).
<i>Toxoplasmosis</i>	Dr. I. A. B. Cathie (E)* and Dr. J. A. Dudgeon (E).
<i>Trichinosis</i> , examination of rats or pigs	Prof. J. J. C. Buckley (E).
<i>Tubercle bacilli</i> , typing	Dr. A. Q. Wells (E).
<i>Typhus Fever</i> , serological tests	Lt.-Col. H. J. Bensted (E).
<i>Note:</i> The Weil-Felix test can be carried out in all constituent laboratories of the Service and also in a number of hospital laboratories. Only sera giving a doubtful reaction should be sent to Lt.-Col. H. J. Bensted.	
<i>Venereal Diseases</i>	Dr. I. N. Orpwood Price (C).
<i>Viruses</i> , material from infections other than those mentioned .•.	Dr. F. O. MacCallum (C).

VACCINES AND SERA OBTAINABLE THROUGH THE PUBLIC HEALTH LABORATORY SERVICE

(These reagents are issued free of charge to medical officers of health and to general practitioners taking part in Local Health Authorities' arrangements made under Section 26 of the National Health Service Act)

Laboratory (For address see list A or B above)	Smallpox Vaccine	Typhus Vaccine	Rabies Vaccine	Anthrax (3) Antiserum
Birmingham (A)	+	+	-	-
Bradford (A) ...	-	-	-	+
Bristol (B) ...	+	+	-	-
Cambridge (A)	+	+	-	-
Carlisle (A) ...	+	-	-	-
Derby (B) ...	+	-	-	-
Dorchester (A)	+	-	-	-
Exeter (A) ...	+	+	-	-
Hereford (A) ...	+	-	-	-
Hull (A) ...	-	-	-	+
Ipswich (A) ...	+	-	-	-
Leeds (A) ...	+	+	-	-
Leicester (A) ...	+	-	-	-
Lincoln (A) ...	+	-	-	-
Liverpool (A) ...	+	+	+	+
London (Colin- dale) (A) ...	+	+	+	+
Maidstone (A) ...	+	-	-	-
Manchester (A)	+	+	-	-
Newcastle (A)	+	+	+	+
Northallerton (A)	+	-	-	-
Northampton (A)	+	-	-	+
Norwich (A) ..	+	-	-	-
Oxford (A) ...	+	+	-	-
Preston*	+	-	-	-
Sheffield (A) ...	+	+	-	-
Taunton (A) ..	-	-	-	+
Wakefield (A) ...	+	-	-	-
Winchester (A)	+	-	-	-
Worcester (A) ...	-	-	-	+
Cardiff (A) ...	+	+	+	+
Carmarthen (A)	+	-	-	-
Conway (A) ...	+	-	-	-

+ means that a stock is normally held.

-- means that a stock is not normally held.

* Pathological Laboratory, Preston Royal Infirmary, Tel. Preston 4616.
Dr. A. A. Miller.

NOTE 1.—*Diphtheria prophylactics* may be obtained by local authorities through any Constituent or Associated laboratory of the Service.

2.—The Central Public Health Laboratory at Colindale, Constituent and most Associated laboratories have stocks of *gamma globulin* for issue under strictly defined conditions.

- NOTE 3.—For *prophylactic* use only. For list of hospitals from which anti-serum may be obtained for treatment, see pages 619 and 620.
- „ 4.—A list of centres where inoculation against *yellow fever* can be carried out is obtainable from the Ministry of Health, Savile Row, London, W.1. (Tel. REGent 8411).
- „ 5.—T.A.B.C., *cholera* and most other vaccines are available commercially.
- „ 6.—Material for *intradermal diagnostic tests*: Frei antigen for Lymphogranuloma inguinale, Brucellin for Undulant fever, Trichina antigen for Trichinosis and Hydatid antigen for Hydatid disease can be obtained from Lt.-Col. H. J. Bensted, Standards Laboratory for Serological Reagents, Central Public Health Laboratory, Colindale Avenue, London, N.W.9 (Tel.: Colindale 7041). Coccidioidin, histoplasmin and Blastomycosis antigen for diagnostic skin tests may be obtained from the Dept. of Pathology, St. John's Hospital for Diseases of the Skin, Lisle Street, London, W.C.2 (Tel.: Gerrard 8383, Ext. 22).
- „ 7.—Certain reagents for the diagnosis of venereal disease can be obtained on payment from Dr. I. N. Orpwood Price at the Venereal Diseases Reference Laboratory, "X" Block, Eastern Hospital, Homerton Grove, London, E.9 (Tel.: Amherst 1532).

Commercial suppliers of calf lymph in an emergency are:—

LIVERPOOL—Evans Medical Supplies Ltd., Speke, Liverpool 19. Tel. HUNts Cross 1881.

After 5.30 p.m. and at week-ends apply to Mr. W. P. Brimage, 61 Hale Road, Liverpool 19. Tel. HUNts Cross 1091.

LONDON—John Bell and Croyden, 50 Wigmore Street, London, W.1. Tel. WEIbeck 5555.

William Martindale, 75 New Cavendish Street, London, W.1. Tel. LANgham 2441.

Allen and Hanburys, 7 Vere Street, London, W.1. Tel. GROsvenor 7571. (Closed at 6 p.m. on weekdays and at 1 p.m. on Saturdays).

Jenner Institute for Calf Lymph, 73-77 Battersea Church Road, London, S.W.11. Tel. BATtersea 1347.

EDINBURGH—T. and T. Smith Ltd., Blandfield Chemical Works, Wheatfield Road, Edinburgh 11. Tel. Edinburgh 63922.

There are no commercial suppliers in Northern Ireland.

SCOTLAND

In Scotland supplies of anti-leptospiral serum, anti-anthrax serum, typhus vaccine, snake anti-venom (for adder bites), rabies vaccine and botulinus anti-toxin are held at The Bacteriological Laboratory, University of Edinburgh, Teviot Place, Edinburgh 1 (Tel. 41001). After laboratory hours telephone Dr. Helen Wright 63711, or Dr. R. R. Gillies, Loanhead 219. Supplies are also held at King's Cross Hospital, Dundee (Tel. 85241).

Tetanus anti-toxin, T.A.B.C., gas gangrene anti-toxin, tetanus toxoid, dysentery vaccine and cholera vaccine should be obtained in Scotland through ordinary trade services.

Vaccine lymph and diphtheria prophylactic can be obtained from:—Bridge of Earn Hospital, Bridge of Earn, Perthshire (Tel. Bridge of Earn 331).

Law Junction Hospital, Carlisle, Lanarkshire (Tel. Wishaw 621).

Peel Hospital, near Galashiels, Selkirkshire (Tel. Galashiels 2295).

NORTHERN IRELAND

In Northern Ireland The Royal Victoria Hospital, Belfast (Tel. 30503) holds supplies of anti-thorax serum, botulism anti-toxin and gas gangrene anti-toxin (but not anti-leptospiral serum). The Central Laboratory of the Northern Ireland Hospitals Authority, 51 Lisburn Road, Belfast (Tel. 28455) holds stocks of anti-anthrax serum, botulism anti-toxin, vaccine lymph, anti-rabies vaccine, typhus vaccine and gamma globulin. Typhus vaccine is also kept at the Belfast City Hospital, 51 Lisburn Road, Belfast (Tel. 29241). The Public Health Laboratory, Water Office, Belfast (Tel. 22731) also stocks vaccine lymph. Snake-bite serum, anti-phallinic serum and anti-leptospiral serum are not kept in Northern Ireland.

EIRE

In Eire vaccines and sera may be obtained from drug firms and pharmacists. Vaccine lymph may also be obtained from the National Vaccine Institute, 80 Sandymount Road, Dublin (Telephone 63622). A small supply of rabies vaccine is held by the Director of the Vaccine Branch of the Department of Health at University College, Earlsfort Terrace, Dublin (Telephone 52116). The advice of the local Chief Medical Officer or of the Medical Officer in charge of the nearest Fever Hospital could be sought in any case of doubt or difficulty.

CENTRES FOR FREE YELLOW FEVER VACCINATION

IMPORTANT.—(1) Every person requiring vaccination MUST MAKE AN APPOINTMENT WITH THE CENTRE: where times of attendance are shown, they are given FOR GUIDANCE ONLY.

(2) No other vaccination is done at these Centres except those marked *.

Town	Address	Tel. No.	Time of attendance (see note (1)* above)
ENGLAND AND WALES			
London	*Hospital for Tropical Diseases, 4 St. Pancras Way, London, N.W.1.	Euston 6441 Ext. 137	<i>Yellow fever vac- cination:</i> Monday, Wednesday and Friday, 11.30 a.m. to 12.30 p.m. Tuesday and Thursday 12.30 to 1.15 p.m. <i>Other vaccinations:</i> Monday, Wednesday and Friday 12.30 to 1.15 p.m. Tuesday and Thursday 11.30 a.m. to 12.30 p.m. Monday to Friday 9 a.m. to 5.15 p.m. Saturday 9 a.m. to 12.30 p.m.
"	*British Overseas Airways Corporation Medical Department, Airways Terminal, Buckingham Palace Road, Victoria, S.W.1. (A charge is made at this centre).	Victoria 2323	Tuesday and Friday 3.30 to 4.30 p.m.
"	Unilever Ltd., Medical Department, Unilever House, Blackfriars, London, E.C.4.	Central 7474 Ext. 91	Monday, Tuesday and Friday 11 a.m. to noon
"	*West London Designated Vaccinating Centre, 53 Great Cumberland Place, London, W.1.	Ambassador 6456	Tuesday, Wednesday and Thursday 2 to 3 p.m.
Birmingham	14 Hagley Road, Edgbaston, Birmingham, 15.	Edgbaston 3861/6	Tuesday 2 to 3.30 p.m.
Bournemouth	Royal Victoria Hospital, Shelley Road, Boscombe, Bournemouth.	Boscombe 35201/8	Tuesday 3 p.m.
Brighton	Royal Sussex Hospital, Pathological Department (Stephen Ralli Memorial), Eastern Road, Brighton, 7.	Brighton 21616	By appointment only

Town	Address	Tel. No.	Time of attendance
ENGLAND AND WALES			
Bristol ...	Regional Transfusion Centre, Southmead, Bristol.	Bristol 628021/5	Tuesday 2.15 p.m.
Cambridge ...	Regional Transfusion Centre, Brooklands Avenue, Cambridge.	Cambridge 56912	Monday 2.30 to 3.30 p.m.
Cardiff ...	Regional Transfusion Centre, 19 Newport Road, Cardiff.	Cardiff 28356	Monday 2.30 p.m.
Hull ...	Hull Royal Infirmary, Prospect Street, Hull.	Hull 36991	Monday 10 a.m. Friday 10.30 a.m.
Kingston-on-Thames ...	*Kingston Group Pathological Laboratory, 37 Coombe Road, Kingston-on-Thames.	Kingston 9844	By appointment only
Leeds ...	Regional Transfusion Centre, Bridle Path, York Road, Seacroft, Leeds, 8.	Leeds 64-5091	Tuesday and Friday 2 to 2.30 p.m.
Liverpool ...	34 Oxford Street, Liverpool, 7. (<i>All communications to be sent to—102 Whitechapel, Liverpool, 1</i>).	Liverpool Royal 6314	Tuesday 2.30 to 3.30 p.m.
Manchester ...	Regional Transfusion Centre, Roby Street, Manchester, 1.	Manchester Central 8181	Tuesday 3 p.m. prompt.
Middlesbrough	Central Clinical Laboratory, General Hospital, Ayresome Green Lane, Middlesbrough.	Middlesbrough 87791	By appointment only
Newcastle-on-Tyne	Regional Transfusion Centre, 78 Jesmond Road, Newcastle-on-Tyne, 2.	Newcastle 81-2271/3	Monday 2 p.m. prompt.
Oxford ...	Regional Transfusion Centre, Churchill Hospital, Headington, Oxford.	Oxford 61361	Monday 2 to 2.30 p.m.
Plymouth ...	Regional Transfusion Sub-Centre, South Devon and East Cornwall Hospital, Freedom Fields, Plymouth.	Plymouth 62228	By appointment only
Sheffield ...	Fargate House (basement), Fargate, Sheffield 1.	Sheffield 63271	Wednesday 3 p.m.
Southampton	Royal South Hants Hospital, Pathological Laboratory, Exmoor Road, Southampton.	Southampton 26211 Ext. 46	Tuesday 2.30 p.m.
Truro ...	Royal Cornwall Infirmary, Truro, Cornwall.	Truro 3029	Tuesday 11 a.m.

Town	Address	Tel. No	Time of attendance
SCOTLAND			
Aberdeen ...	Regional Laboratory, City Hospital, Urquhart Road, Aberdeen.	Aberdeen 22242 Ext. 11	Thursday 2.15 p.m.
Dundee ..	University of St. Andrews, Bacteriological Department, Medical School, 60 Small's Wynd, Dundee.	Dundee 2144	Monday 2 p.m.
Edinburgh ...	Tropical Diseases Unit, Eastern General Hospital, Seafield Street, Leith.	Edinburgh 35463 Ext. 31	Monday and Wednesday 2 to 3 p.m.
Glasgow ...	Public Health Clinic, 20 Cochran Street, Glasgow, C.1.	Glasgow Central 9600 Ext. 302	Friday 2.30 p.m.
NORTHERN IRELAND			
Belfast ..	Belfast City Hospital, Lisburn Road, Belfast.	Belfast 29241	Thursday 9.30 a.m.
EIRE (Inoculation is not free of charge in Eire)			
Dublin ...	Professor F. S. Stewart, Department of Bacteriology, Trinity College, Dublin.	Dublin 66565 (Private number Dublin 63227)	
Ennis ...	Dr. G. P. McCarthy, County Medical Officer, Ennis, Co. Clare.	Ennis 254 (Private number Ennis 111)	
Shannon Airport ...	Dr. William Flynn, Shannon Airport, Co. Clare.	Shannon Airport 207	

Appendix II

HÆMOPHILIA

A hæmophilic should carry a green explanatory card issued jointly by The Medical Research Council, The Ministry of Health and The Department of Health for Scotland. This informs the doctor of the clotting defect and thus protects the patient against the risks of operation. It also names the hospital to which the patient should be referred in an emergency.

The following is the current list of reference centres for patients suffering from hæmophilia and closely related diseases (*e.g.*, Christmas disease).

Aberdeen.

Professor H. W. Fullerton, M.D., F.R.C.P., The Department of Medicine, Foresterhill, Aberdeen. Aberdeen 26341, Ext. 54.

Birmingham.

M. J. Meynell, Esq., M.D., M.R.C.P., D.P.H., The United Birmingham Hospitals, The General Hospital, Birmingham, 4. Central 8611.

Bristol.

Department of Medicine, The Royal Infirmary, Bristol. Bristol 22041.

Cambridge.

F. G. J. Hayhoe, Esq., M.D., Hæmatology Clinic, Department of Medicine, Tennis Court Road, Cambridge. Cambridge 58234/5.

Cardiff.

Professor H. Scarborough, M.B., F.R.C.P.E., F.R.S.E., Medical Unit, Royal Infirmary, Cardiff. Cardiff 25301.

Dundee.

Professor R. B. Hunter, M.B.E., M.B., F.R.C.P.E., Department of Pharmacology and Therapeutics, Medical School, Queen's College, Dundee. Dundee 3204.

Edinburgh.

Sir Stanley Davidson, M.D., F.R.C.P.E., F.R.C.P., F.R.S.E., The Royal Infirmary, Lauriston Place, Edinburgh, 3. FOuntainbridge 2477.

Exeter.

J. O. P. Edgcumbe, Esq., M.D., Department of Pathology, Royal Devon and Exeter Hospital, Exeter. Exeter 2261.

Glasgow.

Professor L. J. Davis, M.D., F.R.C.P., F.R.F.P.S., F.R.S.E.,
Department of Medicine, Royal Infirmary, Glasgow. Bell 3535.

London.

Dr. I. A. B. Cathie, The Hospital for Sick Children, Great Ormond
Street, W.C.1. Holborn 9200.

J. V. Dacie, Esq., M.D., M.R.C.P., Postgraduate Medical School of
London, Duane Road, London, W.12. SHEpherds Bush 1260.

Dr. W. M. Davidson, Department of Clinical Pathology, King's
College Hospital, London, S.E.5. Brixton 6222.

Dr. R. M. Hardisty, Louis Jenner Laboratory, St. Thomas's Hospital,
London, S.E.1. WATERloo 5656.

Dr. C. A. Holman, Lewisham Group Laboratory, Lewisham Hospital,
London, S.E.13. Lea Green 5631.

Manchester.

J. F. Wilkinson, Esq., M.D., F.R.C.P., F.R.I.C., Department of
Haematology, The Royal Infirmary, Manchester, 13. ARDwick 3300.

Newcastle.

C. C. Ungley, Esq., M.D., F.R.C.P., Royal Victoria Infirmary, Queen
Victoria Road, Newcastle-upon-Tyne, 1. Newcastle 25131.

Oxford.

R. G. Macfarlane, Esq., M.D., The United Oxford Hospitals, The
Radcliffe Infirmary, Oxford. Oxford 48481.

Sheffield.

E. K. Blackburn, Esq., M.D., F.R.F.P.S., The United Sheffield
Hospitals, Department of Haematology, The Royal Infirmary,
Sheffield, 6. Sheffield 20161.

In addition to the official centres the following hospitals in London
have special facilities for hæmophiles:—

St. Bartholomew's Hospital, London, E.C.1. (Dr. R. Bodley Scott).
MONarch 7777.

St. George's Hospital, London, S.W.1. (Dr. J. L. Stafford).
SLOane 7151.

The London Hospital, Turner Street, London, E.1. (Dr. H. B. May).
BIShopsgate 3255.

Help other than medical treatment may also be obtained through:—
The Hæmophilia Society, 94 Southwark Bridge Road, London, S.E.1.
WATERloo 3007.

Appendix III

SPECIAL CENTRES FOR POLIOMYELITIS

Arrangements for the provision of a portable respirator for use during the journey should be made with the hospital concerned.

Scotland.

Northern Region (Inverness).

Culduthel Hospital, Inverness. Tel. 2653.

North Eastern Region (Aberdeen).

City Hospital, Urquhart Road, Aberdeen. Tel. 22242.

Eastern Region (Dundee).

King's Cross Hospital, Dundee. Tel. 85241.

South Eastern Region (Edinburgh).

City Infectious Diseases Hospital, Greerbank Drive, Edinburgh, 10
Tel. Edinburgh 51001.

Western Region (Glasgow).

Ruchill Infectious Diseases Hospital, Bilsland Drive, Glasgow, N.W.
Tel. Maryhill 3232.

Belvidere Infectious Diseases Hospital, London Road, Glasgow, E.1.
Tel. Bridgeton 2322.

England.

Newcastle Region.

Walker Gate Hospital, Newcastle upon Tyne, 6. Tel. Newcastle 55131.

Sheriff Hill Hospital, Gateshead, 9. Tel. Low Fell 76787.

Havelock Hospital, Sunderland. Tel. Sunderland 4462.

West Lane Hospital, Middlesbrough. Tel. Middlesbrough 87736.

Isolation Block, Cumberland Infirmary, Carlisle. Tel. Carlisle 22332.

Darlington Memorial Hospital (Hundens Unit) Darlington. Tel.
Darlington 2793.

Leeds Region.

Leeds Road Fever Hospital, Bradford. Tel. Bradford 27125.

Yearsley Bridge Hospital, York. Tel. York 3438.

Seacroft Hospital, York Road, Leeds. Tel. Leeds 648164

Castle Hill Hospital, Cottingham, Nr. Hull. Tel. Cottingham 47372/3.

Manchester Region

Monsall Hospital, Monsall Road, Newton Heath, Manchester, 10
Tel. Collyhurst 2254.

Isolation Hospital, Deepdale Road, Preston. Tel. Preston 5291.

Cherry Tree Hospital, Cherry Tree Lane, Stockport. Tel. Stepping
Hill 2449.

Liverpool Region.

Fazakerley Hospital, Longmoor Lane, Liverpool, 9. Tel. Aintree 2324.

Sheffield Region.

Kendray Infectious Diseases Hospital, Barnsley. Tel. Barnsley 4057.

Lodge Moor Infectious Diseases Hospital, Sheffield. Tel. Sheffield
31124/7

Doncaster Infectious Diseases Hospital, Doncaster. Tel. Doncaster
53241.

Springfield Hospital, Scartho, Grimsby. Tel. Grimsby 7234.
 Lincoln Isolation and Chest Hospital, Lincoln. Tel. Lincoln 8430.
 Leicester Isolation and Chest Hospital, Leicester. Tel. Leicester 20601.
 Heathfield Hospital, Nottingham. Tel. Nottingham 66627.
 Derwent Hospital, Derby. Tel. Derby 42208.

Birmingham Region

Little Bromwich Infectious Diseases Hospital, Birmingham. Tel. Victoria 4021.
 Moxley Infectious Diseases Hospital, Wednesbury. Tel. Wednesbury 0754.
 Whitley Infectious Diseases Hospital, Coventry. Tel. Coventry 2172.
 Bucknall Infectious Diseases Hospital, Stoke-on-Trent. Tel. Stoke-on-Trent 25191.
 Copthorne (Infectious Diseases Wing) Hospital, Shrewsbury. Tel. Shrewsbury 4641.
 Monkmoor Infectious Diseases Hospital, Shrewsbury. Tel. Shrewsbury 6139.
 County Hospital, Hereford. Tel. Hereford 4201.
 Newtown Infectious Diseases Hospital, Worcester. Tel. Worcester 3080.
 Isolation Hospital, Wolverhampton. Tel. Wolverhampton 20008.
 Hayley Green Hospital, Halesowen, near Birmingham. Tel. Halesowen 1002.

East Anglian Region.

Brookfields Hospital, Mill Road, Cambridge. Tel. Cambridge 87132.
 St. Helen's Hospital, Foxhall Road, Ipswich. Tel. Ipswich 77211.
 Norwich Isolation Hospital, Bowthorpe Road, Norwich. Tel. Norwich 25112.
 Great Yarmouth Isolation Hospital, Estcourt Road, Great Yarmouth. Tel. Great Yarmouth 3951.
 Peterborough Isolation Hospital, Fengate, Peterborough. Tel. Peterborough 3134.

Oxford Region.

Stoke Mandeville Hospital, Mandeville Road, Aylesbury. Tel. Aylesbury 900.
 St. Margaret's Hospital, Stratton Street, Margaret, Swindon. Tel. Swindon 2291.
 Harborough Road Hospital, Northampton. Tel. Northampton 1085.
 The Slade Hospital, Oxford. Tel. Oxford 77175.
 Prospect Park Hospital, Reading. Tel. Reading 54827.

South Western Region.

Ham Green Hospital, Bristol. Tel. Bristol 31165.
 The Scott Isolation Hospital, Plymouth. Tel. Plymouth 64311 and 61437.
 Bath Isolation Hospital, Claverton Down, Bath. Tel. Coombe Down 3278.
 Taunton Isolation Hospital, Cheddon Road, Taunton. Tel. Taunton 2396.
 The Isolation Hospital, Whipton, Exeter. Tel. Exeter 67158.
 The Isolation Hospital, Truro. Tel. Truro 3071.

North East Metropolitan Region.

Rush Green Hospital, Rush Green Road, Romford, Essex. Tel. Romford 7711.
 St. Ann's Hospital, St. Ann's Road, Tottenham, N.15. Tel. Stamford Hill 0121.
 Eastern Hospital, Hemerton Grove, London, E.9. Tel. Amherst 1193.
 (Limited availability pending completion of new unit).

North West Metropolitan Region.

South Middlesex Hospital, Mogden Lane, Isleworth, Middlesex. Tel. Popesgrove 2841.

Neasden Hospital, Brentfield Road, London, N.W.10. Tel. Willesden 1850.

North Western Wing of Royal Free Hospital, Lawn Road, Hampstead, N.W.3. Tel. Primrose 7671.

South East Metropolitan Region.

Hither Green Hospital, Lewisham. Tel. Hither Green 3481.

The Brook Hospital, Woolwich. Tel. Woolwich 1172.

Joyce Green Hospital, Dartford. Tel. Dartford 3231.

Pembury Hospital, Tunbridge Wells. Tel. Pembury 263.

Haine Hospital, Isle of Thanet. Tel. Thanet 61511.

Foredown Hospital, Brighton. Tel. Hove 48378.

South West Metropolitan Region.

Western Hospital, Seagrave Road, Fulham, S.W.6. Tel. Fulham 2105.

Infectious Diseases Hospital, Milton Road, Portsmouth. Tel. Portsmouth 2046.

Chest Hospital, Oakley Road, Millbrook, Southampton. Tel. Southampton 71042.

Alderney I.D. Hospital, Ringwood Road, Parkstone, Poole, Dorset. Tel. Parkstone 21.

Victoria Hospital, Alresford Road, Winchester. Tel. Winchester 2048.

Odstock Hospital, Fisherton Street, Salisbury, Wilts. Tel. Salisbury 5111.

Fairlee Hospital, Newport, Isle of Wight. Tel. Newport 2106.

West Haven Hospital, Radipole Lane, Weymouth, Dorset. Tel. Weymouth 16.

Wales.

City Isolation Hospital, Cardiff. Tel. Cardiff 21466.

Hill House Isolation Hospital, Swansea. Tel. Swansea 23551.

Isolation Branch, Maelor General Hospital, Wrexham. Tel. Wrexham 3027.

Galltysil Hospital, Nr. Caernarvon. Tel. Caernarvon 8.

Northern Ireland.

The Northern Ireland Fever Hospital, Belfast. Tel. Belfast 42942.

Isle of Man.

Nobles Hospital, Douglas. Tel. Douglas 987.

Eire. No special centres.

Appendix IV

NIGHT BLOOD BANKS

Many hospitals holding stocks of blood can supply it in an emergency by night as well as by day. In some places application for blood at night should be made to a special hospital as follows:—

ENGLAND

Amersham.

Amersham General Hospital. Tel. Amersham 1351.

Aylesbury.

Royal Buckinghamshire Hospital. Tel. Aylesbury 1420.

Stoke Mandeville Hospital. Tel. Aylesbury 900.

Tindal General Hospital. Tel. Aylesbury 767.

Banbury.

Horton General Hospital. Tel. Banbury 2261.

Barnet.

Barnet General Hospital. Tel. Barnet 7241.

Barnstaple.

Pathological Laboratory, Boutport Street, Barnstaple. Tel. Barnstaple 2322.

Bath.

Department of Pathology, St. Martin's Hospital, Combe Down, Bath. Tel. Combe Down 2383.

Bristol.

Bristol Royal Hospital, Royal Infirmary Branch, Upper Maudlin Street, Bristol, 2. Tel. Bristol 2-2041.

Cambridge.

Regional Blood Transfusion Centre, Brooklands Avenue, Cambridge. Tel. Cambridge 56912. (Mon. to Fri. 8.30 a.m. to 5.30 p.m., Sat. 8.30 a.m. to 12.30 p.m.). Cambridge 56913 at all other times.

Chelmsford.

St. John's Hospital. Tel. Chelmsford 4467.

Cheltenham.

General, Eye and Children's Hospital, Cheltenham. Tel. Cheltenham 2016.

Edgware.

Edgware General Hospital. Tel. Edgware 2381.

Exeter.

Department of Pathology, Royal Devon and Exeter Hospital, Exeter. Tel. Exeter 3549.

Gloucester.

Department of Pathology, Gloucester Royal Hospital, Southgate Street, Gloucester. Tel. Gloucester 23584.

Harold Wood.

Harold Wood Hospital, Essex. Tel. Ingrebourne 2881.

High Wycombe.

High Wycombe and District War Memorial Hospital. Tel. High Wycombe 2464.

Kettering.

Kettering General Hospital. Tel. Kettering 4441.

Liverpool.

Regional Blood Transfusion Centre, Whitechapel, Liverpool, 1. Tel. Royal 6314.

London.

The London Hospital. Tel. BISHopsgate 3255.

Paddington General Hospital. Tel. CUNningham 4884.

Maidstone.

S. London Regional Blood Transfusion Centre, Weaving House, Ashford Road, Maidstone. Tel. Maidstone 4660 and 3718.

Manchester.

Regional Blood Transfusion Centre, Roby Street, Manchester, 1. Tel. CENTral 8181.

Marlborough.

Severnake Hospital. Tel. Marlborough 591.

Northampton.

Northampton General Hospital. Tel. Northampton 4680.

Oxford.

Churchill Hospital. Tel. Oxford 48651.

Radcliffe Infirmary. Tel. Oxford 48481.

Penzance.

West Cornwall Hospital. Tel. Penzance 2382.

Plymouth.

Regional Transfusion Sub-centre, South Devon and East Cornwall Hospital, Freedom Fields, Plymouth. Tel. Plymouth 62228.

Reading.

Royal Berkshire Hospital. Tel. Reading 81721.

Redruth.

Canborne - Redruth Miner's and General Hospital. Tel. Redruth 411.

Southmead.

Regional Transfusion Centre, Southmead. Tel. Bristol 62-8021-3.

Swindon.

Victoria Hospital. Tel. Swindon 4141.

Taunton.

Area Laboratory, Musgrove Park Hospital, Taunton. Tel. Taunton 3662.

Torquay.

Pathological Department, Torbay Hospital, Torquay. Tel. Torquay 63112 Day, 62354 Night.

Truro.

Pathological Department, Royal Cornwall Infirmary, Truro. Tel. Truro 3081 and 2212.

Weston-Super-Mare.

Department of Pathology, General Hospital, Weston-Super-Mare. Tel. Weston-Super-Mare 4321.

Yeovil.

Yeovil Hospital, Higher Kingston, Yeovil. Tel: Yeovil 1671.

ISLE OF MAN

Douglas.

Nobles Hospital, Douglas. Tel. Douglas 3303.

SCOTLAND

Aberdeen.

Royal Infirmary. Tel. Aberdeen 26341.

Dundee.

Royal Infirmary. Tel. Dundee 3125.

Edinburgh.

Blood Transfusion Laboratory, Edinburgh Royal Infirmary, Edinburgh, 3. Tel. FOUNTAINBRIDGE 2477.

NORTHERN IRELAND

Belfast.

Royal Victoria Hospital. Tel. Belfast 30503.

WALES

Aberystwyth.

Aberystwyth General Hospital. Tel. Aberystwyth 374.

Bangor.

Carnarvon and Anglesey General Hospital. Tel. Bangor 1181.

Bridgend.

Bridgend General Hospital. Tel. Bridgend 1016.

Cardiff.

Llandough Hospital, Penarth, Nr. Cardiff, Glam. Tel. Penarth 1602.
Royal Infirmary, Cardiff. Tel. Cardiff 33101.
St. David's Hospital, Cardiff. Tel. Cardiff 20441.

Carmarthen.

West Wales General Hospital, Glangwili, Carmarthen. Tel. Carmarthen 6733.

Chepstow.

St. Lawrence Hospital, Chepstow, Mon. Tel. Chepstow 2334.

Church Village.

East Glamorgan Hospital, Church Village, Nr. Pontypridd, Glam. Tel. Newton Llantwit 86.

Griffithstown.

County Hospital, Griffithstown, Mon. Tel. Griffithstown 261.

Haverfordwest.

County War Memorial Hospital, Haverfordwest, Pembs. Tel. Haverfordwest 714.

Llanelli.

Llanelli General Hospital, Llanelli, Carms. Tel. Llanelli 1313.

Merthyr.

St. Tydfil's Hospital, Merthyr Tydfil, Glam. Tel. Merthyr 900.

Neath.

Neath General Hospital, Neath, Glam. Tel. Neath 2001.

Newport.

Royal Gwent Hospital, Newport, Mon. Tel. Newport 63944.
St. Woolos Hospital, Stow Hill, Newport, Mon. Tel. Newport 63281.

Pontypool.

Pontypool and District Hospital, Pontypool, Mon. Tel. Pontypool 611.

Rhyl.

Group Pathology Laboratory, Royal Alexandra Hospital, Rhyl. Tel. Rhyl 1640.

St. Athan.

R.A.F. Hospital, St. Athan, Glam. St. Athan 55.

Sully.

Sully Hospital, Sully, Glam. Sully 66.

Swansea.

Beck Laboratory, Swansea General Hospital, Swansea. Tel. Swansea 2001.

Morrison Hospital, Morrison, Swansea. Tel. Swansea 7221.

Tredeggar.

St. James' Hospital, Tredeggar, Mon. Tredeggar 371.

Wrexham.

Group Pathology Laboratory, Maelor General Hospital, Wrexham. Tel. Wrexham 3027.

Pathology Laboratory, War Memorial Hospital, Wrexham. Tel. Wrexham 2284.

Appendix V

THE EMERGENCY BED SERVICE (E.B.S.)

If a doctor cannot secure a bed for an urgent case he may ask the E.B.S. to find one. In the Metropolitan Police District this is done by telephoning HOP 7181 (The E.B.S., Fielden House, 28 London Bridge Street, London, S.E.1) at any hour. All acute cases except those of mental disease and tuberculosis (*i.e.*, all cases suitable for general hospitals) will be dealt with. After office hours requests concerning emergencies in tuberculous patients will be accepted. Beds are not found for epileptics except those in status epilepticus. Admission of cases of urgent mental illness should be arranged through the "duty authorised officer" (Mental Welfare Officer) (*see page 226*). Cases of sudden illness or accident occurring in a "public place" in the street, at work or in a doctor's surgery, but not in the home, will be taken to the nearest hospital if an ambulance is requested by dialling 999 in London (or 0 or 01 in other districts). Patients already under treatment should not be sent to hospital in this way. The E.B.S. can often provide useful information on various services needed in emergency situations.

Emergency Bed Bureaux exist in many other centres, some of which are:—

Bromley.

Lennard Hospital, Lennard Road, Bromley, Kent. Tel. Hurstway 2195.

9.0 a.m.-6.0 p.m. seven days a week. Usually open Bank Holidays and Christmas Day.

Chelmsford.

St. John's Hospital; Wood Street, Chelmsford, Essex. Tel. Chelmsford 4851.

8.0 a.m.-8.0 p.m. seven days a week including Bank Holidays and Christmas Day

Liverpool.

The Liverpool Emergency Bed Bureau, 19 James Street, Liverpool, 2. Tel. CENTral 9431.

Luton.

Luton and Dunstable Hospital, Dunstable Road, Luton, Bedfordshire. Tel. Luton 1810.

8.0 a.m.-8.0 p.m. Monday-Friday.

8.0 a.m.-1.0 p.m. Saturdays.

8.0 a.m.-6.0 p.m. Bank Holidays.

Closed on Christmas Day.

Redhill.

Redhill County Hospital, Earlswood Common, Redhill, Surrey. Tel. Redhill 3883.

9.0 a.m.-5.0 p.m. seven days a week including Bank Holidays.

Closed on Christmas Day.

Shrewsbury.

Shrewsbury Emergency Bed Bureau, Royal Salop Infirmary, Shrewsbury. Tel. Shrewsbury 2056. (Also special number Shrewsbury 2856).

Watford.

Shrodells Hospital, Vicarage Road, Watford, Hertfordshire. Tel. Watford 5577, Watford 5353.

9.0 a.m.-5.0 p.m. Monday-Friday.

9.0 a.m.-12 noon Saturdays.

Closed on Bank Holidays and Sundays.

Windsor.

King Edward VII Hospital, Windsor, Berkshire. Tel. Windsor 1410.

Edinburgh.

The Emergency Bed Bureau, 11 Drumsheugh Gardens, Edinburgh, 3. Tel. Edinburgh 31727.

Glasgow.

Hospital Admission Department, 23 Minto Street, Glasgow, C.1.

Tel. Central 9600 (9.0 a.m.-5.15 p.m.).

Tel. Central 4348 (5.15 p.m.-9.0 a.m.).

Tel. Central 4348 (12.30 p.m. Saturday until 9.0 a.m. Monday).

Belfast.

There is no Emergency Bed Bureau as such, but the Royal Victoria Hospital (Belfast 30503) and the Belfast City Hospital (Belfast 29241) have direct telephone communication and endeavour to provide accommodation for emergencies. In Northern Ireland generally the first hospital approached is responsible for telephoning adjacent areas for accommodation.

Dublin.

Dublin Hospitals Bureau, 52 Upper Mount Street, Dublin. Tel. Dublin 63941.

Appendix VI

CORNEAL GRAFTING

(For technique of collecting eyes see page 493)

The following list gives the names and telephone numbers of hospitals which may be approached by a doctor whose dying patient wishes to donate his eyes for surgical purposes. Those not mentioned do not have facilities for collecting eyes at present. No Act corresponding to the Corneal Grafting Act has been passed in the Isle of Man.

Scotland.

Aberdeen	The Royal Infirmary	Aberdeen 26341
Dundee	The Royal Infirmary	Dundee 3125
Edinburgh	The Royal Infirmary	FOuntainbridge 2477.
Glasgow	Tenant Memorial Institute (Eye Dept. of Western Infirmary)	Western 8822
	The Ophthalmic Institution	Douglas 7408

Northern Ireland.

Belfast	The Royal Victoria Hospital	Belfast 30503
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Eire.

Dublin	Royal Victoria Eye and Ear Hospital	Dublin 61169 and 62804
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Wales.

Cardiff	The Royal Infirmary	Cardiff 33101
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England.

Birmingham	Birmingham and Midland Eye Hospital	CENtral 6711
Blackburn	Royal Infirmary	Blackburn 7133
Blackpool	Victoria Hospital	Blackpool 23881
Burnley	Victoria Hospital	Burnley 6311
Bradford	Royal Eye and Ear Hospital	Bradford 28241
Bristol	Bristol Eye Hospital	Bristol 25535
Burton-on-Trent	Burton-on-Trent General Hospital	Burton 3334

Cambridge	Addenbrooke Eye Hospital	Cambridge 55671
Coventry	Coventry and Warwickshire Hospital	Coventry 40551
Crewe	Crewe District Memorial Hospital	Crewe 4221
Derby	Derbyshire Royal Infirmary	Derby 44255
East Grinstead	Queen Victoria Hospital	Grimsby 5051
Ipswich	East Suffolk and Ipswich Hospital	Ipswich 51021
Huddersfield	The Royal Infirmary	Huddersfield 2866
Leeds	The General Infirmary	Leeds 32799
Leicester	Leicester Royal Infirmary	Leicester 5011
Liverpool	St. Paul's Eye Hospital	CENTRAL 7794
London	Moorfields, Westminster and Central Eye Hospital	CLERKENWELL 6101
	Lambeth Hospital, S.E.11.	RELiance 3804
	St. Thomas's Hospital, S.E.1	WATERloo 5656
	Guy's Hospital, S.E.1	HOP 3500
	Royal Free Hospital, W.C.1	TERminus 6411
	Charing Cross Hospital, W.C.2	TEMPle Bar 7788
	Westminster Hospital, S.W.1	VICToria 8161
	University College Hospital, W.C.1	EUSTon 5050
	King's College Hospital, S.E.5	BRIXton 6222
Louth	Western Ophthalmic Hospital, N.W.1	PADDington 1871
	Louth County Infirmary	Louth 450
Manchester	The Royal Eye Hospital	ARDwich 1115
Newcastle	Royal Victoria Infirmary	Newcastle 25131
Norwich	Norfolk and Norwich Hospital	Norwich 21311
Nottingham	Nottingham and Midland Eye Infirmary	Nottingham 41043
Oxford	Oxford Eye Hospital	Oxford 2253
Preston	Royal Infirmary, Preston	Preston 4616

Reading	Royal Berkshire Hospital	Reading 2231
Sheffield	Sheffield Royal Infirmary	Sheffield 29161
	Sheffield Royal Hospital	Sheffield 20063
Shrewsbury	Eye, Ear and Throat Hospital	Shrewsbury 3083
Southampton	Southampton Eye Hospital	Southampton 22208
Stockport	Stockport Infirmary	Stockport 4847
Stoke-on-Trent	North Staffordshire Royal Infirmary	Stoke-on-Trent 44161
Wigan	Royal Albert Edward Infirmary	Wigan 2841
Wolverhampton	Wolverhampton and Midland Counties Eye Infirmary	Wolverhampton 23605
Worcester	Worcester City and County Eye Infirmary	Worcester 4017
York	County Hospital	York 25314

Appendix VII

DRUGS MENTIONED IN THE TEXT AND SOME OF THEIR EQUIVALENT PREPARATIONS

Adrenaline.

Injection of Adrenaline B.P. 1 in 1,000 (Tartrate).

Solution of Adrenaline Hydrochloride B.P. 1 in 1,000.

Kadamysin (Zimmermann). Adrenaline with pituitary extract
(formerly called Asthmolysin).

Slowly acting preparations.

Adrenaline in oil (Parke Davis). 1 ml. contains 2 mg. of adrenaline hydrochloride (=30 minims of solution of adrenaline hydrochloride B.P.).

Hyperduric adrenaline (Allen & Hanburys). Adrenaline mucate, dose 3 to 8 minims (0.18 to 0.5 ml.).

Adrenutol (Evans). A solution of adrenaline 1 in 500 and chlorbutol 1 in 500 in water and glycerin (dose 1 ml.).

Adrenaline-like substances (preparations of isopropyl-nor-adrenaline sulphate; approved name = isoprenaline).

Isoprenaline (Boots).

Isupren (Bayer). 20 mg. tablets and

Aleudrin (Lewis laboratories). 1 per cent. spray

Neo-epinine (Burroughs Wellcome). solution.

Neodrenal (Savory & Moore).

Isoprenaline sulphate (Allen & Hanburys). 10 mg. tablets and 1 per cent. spray solution.

Norisodrine (Abbott). 10 per cent. and 25 per cent. powder for oral inhalation.

Levophed (Bayer) (l-noradrenaline).

Adrenal cortex preparations.

GRANULAR EXTRACTS.

Injection of Suprarenal Cortex B.P.C.

Eucortone (Allen & Hanburys).

Cortin (Organon).

Eschatin (Parke Davis).

Supracort (Paines & Byrne).

SYNTHETIC (Deoxycortone acetate).

Cortenil (Bayer).

Cortigen (Richter).

Cortiron (British Schering).

D.O.C.A. (Organon).

Percorten (Ciba).

Syn-cortyl (Roussel).

Amethocaine hydrochloride B.P.

Anethaine (Glaxo).

Butethanol.

Decicain (Bayer).

Pontocaine (Winthrop, New York).

Tetracaine Hydrochloride U.S.P. XII.

Pantocaine (German Bayer Co. Leverkusen).

Aminophylline B.P. (Theophylline with Ethylenediamine).

Cardophyllin (Whiffen). Formerly called Euphyllin.
Genophyllin (Genatosan).

AMINOPHYLLINE COMPOUNDS.

- Etophyllate (Rona).
- Neutraphylline (Continental laboratories).

Carbachol B.P.

Doryl (Merck).
Moryl (Savory & Moore).
Choryl (Pharmaceutical Products).
Carbamed (Medo).

Cortisone Preparations.**Cortisone acetate.**

5 mg. and 25 mg. tablets; 25 mg. per ml. 1/m injections; 1 per cent. eye drops; 1 per cent. eye ointment.

Cortelan (Glaxo).
Cortisyl (Roussel).
Cortistab (Boots).

Hydrocortisone acetate.

25 mg. per ml. intra-articular injection. 1 per cent. eye drops; 1 per cent. eye ointment; 1 and 2.5 per cent. ointment (2 bases).

Efcortelan (Glaxo).
Hydrocortisyl (Roussel).
Hydrocortistab (Boots).
Cortef Acetate (Upjohn).
Cortril (Pfizer).
Hydrocortone Acetate (Merck-Sharp & Dohme).

Hydrocortisone free alcohol.

10 mg. and 20 mg. tablets; 100 mg. per 20 ml. I/V injections (to be diluted); 0.5 and 1.0 per cent. lotion.

Efcortelan (Glaxo).
Hydrocortistab (Boots).
Hydrocortisyl (Roussel).
Cortef (Upjohn).
Cortril (Pfizer).
Hydrocortone (Merck-Sharp & Dohme).

Prednisone (Δ^1 -dehydrocortisone).

5 mg. tablets.
Decortisyl (Roussel).
Deltacortelan (Glaxo).
Deltacortone (Merck-Sharp & Dohme).
Ultracortin (Ciba).

Prednisolone (Δ^1 -dehydro-hydrocortisone).

5 mg. tablets.
Codelcortone (Merck-Sharp & Dohme).
Delta-cortef (Upjohn).
Delta-stab (Boots).
Delta-cortril (Pfizer).

Fluoro-hydrocortisone acetate.

0.1 per cent. lotion; 0.1 per cent. ointment.
Florinef (Squibb).

Diodone B.P.

Perabrodil (Bayer).
Pyelosil (Glaxo).
Pylumbrin (Boots).
Diodrast (Winthrop Stearns).
Uriodone (May & Baker).

Ergotamine tartrate.

Femergin (Sandoz).
Gynergen (Sandoz).
Neo-femergin (Sandoz).

Hexobarbitone sodium B.P.

Cyclonal sodium (May & Baker).
Evipan sodium (Bayer).

Hyaluronidase.

Hyalase (Benger). (Solid in ampoule)
Rondase (Evans). (Solid in vial).
Wydase (Wyeth). (Stabilised solution).

Iodised Oil B.P.

Iodatol (B.D.H.).
Iodatum (U.S.P.).
Iodinol (Martindale).
Iodipin (Martindale, Meick).
Lipiodol (Bengue).
Neo-hydriol (May & Baker).

Leptazol B.P. (Called Metrazol in U.S.A.)

Cardiazol (Knoll).
Phrenazol (Boots).

Mersalyl B.P.

Salyrgan (Bayer).
Merphyllin (Richter).
Related mercurial diuretics are:—
Esidrone (Ciba).
Dilurgen (Richter).
Mercardan (Parke Davis).
Mercloran (Parke Davis).
Novurit (Martindale).
Neptal (May & Baker).
Thiomerin (Wyeth).

Methadone Hydrochloride B.P. (Originally called Miadone. Burroughs Wellcome).

Amidone (original German name).
Methadon (T. H. Smith).
Disephonine.
Dolophine (U.S.A.).
Physeptone (Burroughs Wellcome).

Nikethamide B.P.

Coramine (Ciba).
 Anacardone (B.D.H.).
 Corvotone (Boots).
 Nicamide (Burroughs Wellcome)

Oestrogens.

OILY SOLUTIONS FOR INTRAMUSCULAR USE.

Oestradiol Monobenzoate B.P.
 Dimenformon (Organon).
 Oestroform (British Drug Houses).
 Ovocylin P. (Ciba).
 Progynon B. Oleosum (British Schering).
 Thcelin (Parke Davis).
 Uden (Bayer).

ORAL PREPARATIONS.

Ethinylœstradiol B.P.
 Estigyn (British Drug Houses).
 Ethidol (British Schering).
 Ethinœstryl (Roussel).
 Eticyclin (Ciba).
 Lynoral (Organon).

ORAL PREPARATIONS WITH METHYL TESTOSTERONE ALSO.

Mepilin (British Drug Houses).
 Mixogen (Organon).

SYNTHETIC PREPARATIONS.

Dienœstrol.
 Hexœstrol.
 Stilbœstrol B.P.
 Tace (Riker).

Papaveretum B.P.C. Dose 11 to 22 mg. (gr. $\frac{1}{4}$ to $\frac{1}{2}$).

(Not to be confused with **Papaverine B.P.C.**, dose 0.13 to 0.26 G. (gr. 2 to 4), which has mild hypnotic but marked antispasmodic effects).

Omnopon (Roche).
 Alopon (Allen & Hanburys)
 Opioloid (Richter).
 Opoidine (Macfarlan).
 Pavopin (T. & H. Smith).

Parathyroid preparations.

Parathyroid extract B.P.C. 1934 (powder).
 Injection Parathyroid U.S.P. XIV.
 Parathormone (Lilly).
 Paroidin (Parke Davis & Co.).
 Parathyroid Hormone (Paines & Byrne).
 Extracts also prepared by Armour Laboratories, Oxo Ltd., and Gedeon Richter Ltd.

Pentose nucleotide.

Pentide (Allen & Hanburys).
 Pentnucleotide (Menley & James).
 S.P.N. (Evans Medical Supplies Ltd.).

Pethidine Hydrochloride B.P. (Called Demerol in U.S.A.).

Dolanin.
Dolantal (Bayer).
Meperidine.

Procaine hydrochloride B.F.

Ethocaine.
Kerocaine (Kerfoot).
Novocain (Bayer).
Parsetic (Parke Davis & Co.)
Planocaine (May & Baker).
Sevicaine (Glaxo).

Progesterone B.P. Oily solutions for intramuscular injections (containing 1 to 10 mg. per ampoule).

Proluton (British Schering).
Progestin (Organon), (British Drug Houses).
Progesterone (Burroughs Wellcome).
Lutocyclin (Ciba).
Luteostab (Boots).
Lutogyl (Roussel).
Lutren (Bayer).
Gestone (Paines & Byrne).

TABLETS (5 to 10 mg.) FOR ORAL USE.

Ethisterone B.P.
Gestone (Oral) (Paines & Byrne).
Lutocyclin (Oral) (Organon).
Oraluton. (British Schering).
Progestoral (Organon).

Pyridoxine (Adermin, Vitamin B6).

Benadon (Roche). 20 mg. tablets and 50 mg. ampoules.
Pyrirel (Vitamins).
Vitamin B₆.
Hexa-betalin (Lilly).

Thiopentone sodium B.P.

Pentothal sodium (Abbott).
Intraval sodium (May & Baker)

Vitamin C. (Ascorbic acid B.P.).

Cantan (Bayer).
Celin (Glaxo).
Davitamon C. (Organon)
Redoxon (Roche).

Vitamin B. (Aneurine hydrochloride B.P.).

Betavel (Vitamins Ltd.).
Benerva (Roche).
Berin (Glaxo).
Betalin (Lilly).
Betaxan (Bayer).
Crysto-Vibex (Parke Davis).
Davitamon B. (Organon)
Thiamine chloride (Abbott).
Vitamin B₁.

Vitamin E. (Tocopherol acetate B.P.C.).

Davitamon E. (Organon).
 Ephynal (Roche).
 Fertitol (Vitamins Ltd.).
 Germinol (Paines & Byrne).
 •Phytoferol (British Drug Houses).
 Trigol (Abbott).
 Viteolin capsules (Glaxo).
 Zygon (Squibb).
 Wheat-germoil (Crookes).
 Gelucaps (Bioglan).
 Profecundin (Richter).

Vitamin K and Analogues

Natural.

Korakion (Roche).
 Mephyton (Merck-Sharp & Dohme).

Synthetic. Oil soluble for injection.

Menaphthone B.P.

Menadione U.S.P.

Prokayvit (British Drug Houses).

Davitamon K (Organon).

Vitavel K (Vitamins).

Oil soluble oral.

Acetomenaphthone B.P.

Prokayvit oral (British Drug Houses).

• Davitamon K (Organon)

Vitavel K (Vitamins).

Water soluble.

Vitavel K (Vitamins)

Synkavit (Roche)

Kayvisyn (Parke Davis).

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